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PNEUMONIA

THE

NATURAL HISTORY AND RELATIONS

OF

PNEUMONIA

A CLINICAL STUDY

BY

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PREFACE.

AT various times during the last ten years I have been led to discuss certain questions in reference to pneumonia. Thinking to collect these papers in the belief that, taken together, they might lend each other support, it occurred to me that, with some addition and rearrangement, it would be possible to present the subject in a connected form, where matters of doubt and difference should be made subordinate to the main purpose of describing and illustrating the ordinary features of the disease.

While occupied with this design, an essay upon pneumonia by Dr. Wilson Fox appeared in the third volume of 'Reynolds' System of Medicine.' English readers were thus, for the first time, put in possession of a detailed account, both graphic and precise, of an affection which hitherto, in this country, had only been treated of in brief, along with other pulmonary diseases.

As it was far from my desire to challenge comparison with such a work, its publication might have spared me the task of completing my own. It appeared, however, that the author of this treatise acquiesced in a view of the subject which, rightly or wrongly, I had been engaged in opposing. I felt bound to persevere, therefore, in defence of opinions widely entertained, and now more than ever in need of vindication.

What those opinions are and how maintained I leave it for the book to show. Yet it must not be supposed that the pages which follow are given over to controversy. The discussion of what is and what is not pneumonia may easily lapse into an idle quarrel about words. Pneumonia is just what we agree to make it. It is but a term in itself insignificant, and ready for any use that may be required of it.

A personal bias in regard to the nature and relations of lung inflammation must, indeed, of necessity shape and colour what is here set down, yet my single aim has been to describe what appears without afterthought, and, careless of names, to adopt such natural arrangement as might best suit the material to be provided for.

In a treatise of this kind it would seem superfluous to acknowledge any particular source of help. I have picked up from various quarters whatever seemed suitable to my purpose, and, as has been intimated, am particularly indebted to the work of Dr. Wilson Fox. The liberality of the editors of the 'St. George's Hospital Reports' enables me to reproduce some tables which I first published in their second volume. By a similar kindness on the part of my colleagues at the Hospital for Sick Children I have been granted the use of the valuable records of that institution.

The few woodcuts of the volume (which have been selected to illustrate those features of pneumonia which are least often figured in books) were copied with his usual fidelity by Mr. Collings direct from the specimens.

That my obligations do not end here will be seen from the work itself. The facts or opinions of others are not knowingly quoted without acknowledgment. I mention with especial gratitude the kindness of my colleagues, Dr. Basham and Dr. Fincham, in giving me access to many instructive examples of the disease I was studying occurring in their own practice at the Westminster Hospital.

The account already given of the origin of the book, while it may serve to explain some defects in construction, excludes the plea of haste or insufficient consideration. With many faults which I can see without being able to mend, and omissions, arising not from neglect but ignorance, the work is deliberate and as good as I can make it. Yet it owes so much to the co-operation of others that it is no affectation to say now in dismissing it that I ground its prospect of success upon the consideration that but a small portion is properly my own.

LONDON: *Christmas* 1875.

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CHAPTER I.

PNEUMONIA IN HISTORY.

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Pneumonia a representative disease.

THE history of Pneumonia within the present century reflects the history of medical practice in general during a period of singular restlessness and change. By tracing successive doctrines in reference to this single affection from the time when the genius of Laennec first made the study of it possible, we may follow without break, up to the views of to-day, the several principles and modes of thought which have governed pathology in the same interval. Pneumonia is a representative disease. Discussions as to the nature and results of inflammation have chosen it for their chief illustration, while the effect of antiphlogistic treatment has been condemned or approved upon its evidence. When depletion was most in vogue it was to the lung in inflammation that its methods were most relentlessly applied. When the wisdom of bloodletting began to be questioned, it was resolved to test its efficacy by appealing to the results obtained in pneumonia; and, coming to later times, when disease was first recognised as consisting in an orderly succession of invariable phenomena, it was again with pneumonia that the crucial experiment was made of leaving inflammation to itself.

Thus the history of this disease, if only we can faithfully follow it, would seem to comprise something more than the bare enumeration of a certain set of symptoms, or the barren record of a succession of remedies, vaunted at one moment and abandoned the next. It would be an epitome of the manner in which the generation immediately preceding our own regarded disease in general, of the facts relied on to sanction a system of therapeutics far more peremptory than ours, and of the arguments which have since prevailed to modify, or, more truly, to revolutionise the old methods of dealing with acute disease.

Its distinct and separate place among diseases.

And, while thus interesting and important as the type and pattern of inflammatory affections, pneumonia occupies a place of its own, distinct both from strictly local diseases and from those which affect the system generally. Differing from either, it has something in common with both. Besides the local injury, its general phenomena, exhibited over a wide area involving every function of the body, are so fixed as to time and order of succession as to resemble rather a specific fever than the pyrexia of a merely local inflammation.

Yet most of all is the study of this disease to be commended for the promise that attends it. There is a hope and purpose here, keener and brighter than in those organic affections where the utmost effort of art serves only to lengthen out a life which it can hardly render tolerable. Pneumonia, like fever, may assault and kill those who are in the full vigour of life. It is of the deepest concern, therefore, to the public welfare to be resolved how to meet it, while the issues in life or death may depend, we know not as yet how much, upon the skill and discernment of the physician.

The peri-pneumonia of the ancients

The early history of pneumonia is very obscure. Accounts given by ancient writers are not, in fact, sufficiently precise to make it certain that successive authors are describing the same thing. All that can be said is that a condition which, after the nomenclature of Hippocrates, went by the name of *peripneumonia* occupies a conspicuous place amongst the maladies of old. It is clear that the ancient physicians failed to distinguish between pneumonia and acute pleurisy. Hippocrates, obviously confusing the two, describes the latter as a disease of rapid fatality, exhibiting variously coloured sputa. Even our own Sydenham speaks of the two affections as forming one malady, while Cullen, writing only a century ago, comprehends under the title pneumonia the whole of the inflammations affecting either the viscera of the thorax or the membrane lining the internal surface of that cavity. 'Neither do our diagnostics,' says he, 'serve to ascertain exactly the seat of the disease, nor does the difference in the seat exhibit any considerable variation in the state of the symptoms, or lead to any difference in the method of treatment.' As lately, indeed, as 1792,[1] Jean P. Frank, in contradiction of some indistinct affirmations of Valsalva and Morgagni, maintained that pleurisy and pneumonia must be studied under the common name of pleuro-pneumonia.

confounded with pleurisy.

Early recognition of peri-pneumonia notha.

While thus loosely described, it was yet found necessary at an early period to distinguish the actual disease from its supposed counterfeits. 'True,' 'acute,' 'legitimate,' on the one hand; 'spurious,' 'bastard,' 'illegitimate,' 'false,' on the other, are expressions that have been in use for nearly two centuries.

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[1] In the works of Huxham and Hoffman, some years earlier, the term 'pneumonic fever' is adopted, indicating, as Grisolle supposes, the belief of those authors that this special fever, rather than the local condition accompanying it, is the primary matter.

Pneumonia in the great European epidemics.

Traces of the disease we are to describe may be found scattered about the medical history of Europe from the earliest times. In many of the great epidemics of which accounts are preserved the stress of the sickness has fallen chiefly upon the lungs, and contemporary writers have described these organs as 'inflamed,' and named the disorder accordingly. Thus the plague at Athens, 430 years before Christ, has been represented as a gangrenous pneumonia. The account of Thucydides,[1] however, seems to apply rather to a disease in some respects resembling typhoid fever, yet with special features of its own differing from all known epidemics. The extent and the manner of the lung implication it is very difficult to determine. The chief danger of the disease, and its most frequent termination, was by 'ulceration and excessive diarrhœa, by which the patients were afterwards carried off through mere weakness.'

Epidemics much later in time than this are involved in much the same obscurity. Take, for instance, the Black Death of the fourteenth century, which devastated Asia as well as Europe, and destroyed, as was said, two-thirds of the people, besides beasts, birds, and even fishes.[2] The leading symptoms of this pest, as described in a manuscript found in the library of St. Pierre at Lyons, were—cough, bloody spitting, diarrhœa and vomiting, 'together with buboes, anthrax, and petechiæ.' The learned monk, whose curious parchment, written in verse, was accidentally brought to light, recommends as preventives living in a pure air, following a very moderate rule of life, avoiding cold and damp and fœtid emanations, and 'keeping oneself pure as at the day of

Pneumonic symptoms in the 'Black Death.'

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[1] Book ii. chap. xlix.

[2] Ozanam, vol. iv. p. 76 et seq. The statements that follow in reference to the old epidemics are chiefly from the same authority.

baptism;' advice whose real elevation and enlightenment it needs some acquaintance with the practical medicine of the fourteenth century fully to appreciate.

Upon such meagre information, and in the absence of any trustworthy post-mortem description, we can say no more than that these ancient epidemics resembled most certain prevalent fevers of later date, in which the signs of lung mischief occupy a prominent place, and death discloses engorgement or consolidation of those organs. The earlier part of the sixteenth century was remarkable for a series of epidemics apt, under certain conditions, to assume this character. In connection with the general scarcity and famine which marked the revival of learning, a pestilence of this kind extended over Italy, France, Hungary, and Spain. It is described as a maculated fever, and the discussions of the day had reference to its relationship to the true plague.

The epidemics of the sixteenth century in their relation to pneumonia.

Later, in 1557, while an epidemic resembling typhus was prevailing in France, an offshoot from it arose in Belgium which would seem not unfitly described as epidemic pneumonia. Commencing at the end of September, it was preceded by a violent and very cold north wind, whereupon 'catarrhal affections commenced, followed by vehement cough and fever, pain in the side, and difficult respiration; bloody expectoration occurred on the third day, and death on the fifth to the eighth, unless bleeding was performed on the first or second day.' Practised later, the operation was held to be useless.

A similar sickness occurred early in the seventeenth century. Dyspnœa, cough, (dry at first but afterwards viscous, and, later, rust-coloured or bloody,) sharp pleuritic pains at the onset, and at the end delirium. The disease, so similar in these respects to our pneumonia, was supposed to be decidedly contagious. The post-mortem appearances, given in vague terms, may be variously in-

The leading symptoms of the disease decribed in the seventeenth century.

terpreted by the modern pathologist:—'Fœtid water' in the pericardium; 'a pituitous substance,' bloody and purulent, in the lungs.

An epidemic, still better entitled to be called pneumonia, attacked the garrison of Philisbourg in 1688. It was attributed to long-continued, cold north winds, along with the privations and exposure of camp life. At first, as the account runs, the sufferers were easily cured by bleeding, but soon the malady assumed a markedly epidemic character, and, aided by measles and small-pox, made great ravages, especially among the more dissolute of the soldiers. Its symptoms, shortly described, were 'stitch' and chest oppression, terminating, in the fatal cases, in delirium, or convulsions, or diarrhœa, or all three. The usual duration in these instances was from seven to nine days. There is the exceptional interest attaching to this particular epidemic that the post-mortem condition is given in some detail. The lungs are described as 'actively inflamed and hepatised, in many parts purulent, the chest and pericardium filled with bloody serum, and polypi in the right auricle of the heart.' From this last appearance, and from its high mortality, the epidemic got the name of malignant 'polypous peripneumonia.' It need hardly be said that the description, so far as it goes, closely resembles the pneumonia of to-day.

'Sthenic' and 'asthenic' pneumonia in the eighteenth century.

Coming to the eighteenth century, we find a pulmonary epidemic, contagious and generally fatal, raging in Rome, where to the usual symptoms there is added jaundice. It deserves mention in illustration of the fact that our fathers were not slow to discover the failure of their favourite remedy, phlebotomy. Two epidemics—a sthenic and an asthenic, as we might say—seem to have succeeded each other. In the first, which was 'of inflammatory character,' bleeding was beneficial. In the second (in 1713) gangrene was apt to supervene both in the lungs and other organs, and now the operation of bleeding, useful before, was believed to be fatal. Of the earlier epidemic cold weather was thought to be the exciting cause. It disappeared with the advent of summer, and was again provoked, as was supposed, by a cold north wind and rains, under the form of a 'malignant pleurisy.'

The special symptoms of influenza combined with pneumonia.

It would be profitless, with the scanty materials at hand, to allude to other epidemics supposed to be pneumonic. In circumstances and phenomena they have much in common. Associated almost always with inclement weather, especially prolonged and cold north winds, their history included symptoms which nearly resemble (as in the epidemic of 1750 described by Roulin) the influenzas of the present century. Great prostration and limb pains, sometimes with and sometimes without lateral 'stitch,' concur with rust-coloured spitting and other signs of proper pneumonia; 'the cheek of the affected side being commonly covered with a dark red patch,' and the malady ending by critical sweats and the cessation of fever from the fifth to the ninth day. Such was the epidemic of Flanders in 1756, and of Eplechin in 1776, where the likeness to pneumonia is very striking. Diarrhœa, bilious vomiting, and jaundice are other symptoms of frequent occurrence. And, as with influenza so here, bleeding, in some instances resorted to as the only remedy, was in most regarded as absolutely fatal. Of 95 bled at Berne in 1762, 85 died, while only 10 died of 77 who were not bled.

Bloodletting recognised as injurious.

Post-mortem descriptions at this period.

As has been said, it is rare up to the end of the eighteenth century that any further light is obtained from post-mortem inspection. Such scanty observations as we possess are open to various interpretation and remark. Thus while the lungs are often described as 'gangrenous,' the well-marked features of gangrene, and especially the fœtor, which would hardly have escaped record, do not appear in the living history. Some of the descriptions would serve well enough for grey pneumonia, as where the lung is described as covered with 'an ash-coloured membrane,' and its substance as falling away into putrilage. It is remarkable, however, that the morbid condition, whatever it be, involves the lungs wholly; it is never one lung which is inflamed, or gangrenous, or sphacelated. Often, too, 'gangrene' affects other organs, and especially the liver, while not seldom ulceration or 'gangrenous aphthæ' are found in the intestines.

Pleurisy.

The presence of recent pleurisy, which with us is regarded as a valuable indication of the character of the disease, finds little mention in these earlier records. The reason no doubt is that the visceral pleura was not recognised in those days as distinct from the organ it envelopes. Pleurisy, for them, applied to the costal pleura only, and the word is mostly used to denote serous effusion into the pleural cavity.

Ante-mortem clots and pericarditis.

As regards the heart and pericardium, it is significant to find that some of the epidemics were then named 'polypous,' owing to the frequent presence of 'polypi in the right auricle,' a condition, (assuming ante-mortem blood-clots to be meant,) proper to pneumonia. In other instances, it must be confessed, the language used is ambiguous, as where the heart is 'withered' and diminished in volume, or the pericardium contains pus or 'fœtid water.' Pathological nomenclature, even with us, is shifting and fanciful; the same phrases carry one sense to-day and another to-morrow. But at the present time we have the advantage, that the artist lends his aid to render description more durable than heretofore by lifting it above verbal changes.

Our own influenzas in connec-

It will occur to every reader to contrast these desolating epidemics of so-called pneumonia with the historical in-tion with pneumonic epidemics.

fluenzas of our own country. While the former seemed to threaten almost the extermination of mankind, nothing is more remarkable in connection with the latter than the insignificance of their mortality. Although whole populations were stricken, and the symptoms (including high fever and considerable bodily and mental prostration) were apparently grave, death occurred only as an accident.[1] 'I know not,' says Heberden, speaking of such an epidemic just one hundred years ago, 'who could properly be said to die of it.' Nevertheless it seems highly probable that the very same epidemic which in Europe was exhibiting pneumonic symptoms, and obtaining its name and fatality on that account, extended to England under the milder form of a popular catarrh. Our influenza of 1762, to which dysentery succeeded, an epidemic remarkable, in comparison with those that followed it, for its severity and unusually 'inflammatory type,' was probably only part of the same malady—taking colour and shape from the soil on which it rested—which in the canton of Berne raged with such virulence as to destroy life in four-and-twenty hours.[1] Similarly the influenza which appeared in England in the spring of 1710 was the tail of the destructive pneumonic epidemic in Provence and Languedoc which followed the unequalled severity of the great winter 1708–9.

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[1] The increased mortality of those times arose not from the influenza proper, but from the other diseases which prevailed along with it. Dysentery, diarrhœa, measles and (in the later epidemics) cholera, were its frequent attendants, and mark out those years as generally unhealthy. Yet all epidemics of influenza were not alike in this respect; succeeding influenzas varied considerably, as did different localities during the same epidemic. Thus the tendency to pneumonia, and the consequent mortality, was much greater in 1762 than in 1775. Yet in the latter year, in one city—Chester, inflammation of the lung was 'a frequent and dangerous termination.' In the epidemic of 1782 a single village was conspicuous for the inflammatory character of its symptoms. 'It now and then,' says a report of the College of Physicians, 'degenerated into peripneumony, yet these affections manifested themselves as particular modifications of the epidemic; they either preceded or accompanied some of its characteristic symptoms.' In regard to the influence of locality a remark of Hamilton's may be quoted to the effect that 'influenza partook more of pneumonia in dry and open situations.' With this may be compared an observation of Manetti concerning a pneumonic epidemic in Florence, that it was confined to the most airy parts of the city. See Dr. Theophilus Thompson's 'Epidemic Catarrh,' p. 109 et seq.

Modern epidemics of pneumonia.

In modern times and with fuller knowledge, pneumonia in an epidemic form has ceased to be common, yet we are not without instances. Dr. Hjaltelin, the Inspecting Medical Officer of Iceland, reports a visitation of the kind to his own country in the year 1863.[2]

In Iceland in 1363.

'The winter,' says he, 'was an extremely stormy and rough one in this island. People were still in many places, and especially in the north, newly recovered from the epidemic dominion of the influenza, and it seemed to me quite natural that their respiratory organs were still in a state of great sensibility, which might give occasion to acute pulmonary affections during that extremely rough and stormy winter season.' The author, whose vigorous narrative seems to suit the high latitude from which it comes, proceeds to detail the leading features of eighty cases, of which eight died, four of them being past hope when first seen. In two of these death took place after four days' illness, one of them having 'walked about' the day before the seizure. In both cases extensive hepatisation, lymph-covered pleura, coagula in the right ventricle, and granular section, indicate beyond doubt genuine pneumonia, fatal at an early stage.

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[1] As in the old destructive epidemics, an obsolete terminology makes post-mortem accounts difficult to understand, so in our influenzas the rarity of death makes them hard to find. Yet such evidence as we have goes to prove that influenza, in anatomical respects, nearly resembled the great European epidemics. Dr. Graves has described the appearances met with in the influenza of 1837. In most of the cases examined both lungs were affected, easily breaking down under the fingers, and '*the portion so torn resembled gangrenous lung, except that there was an absence of fœtor;*' a description, it will be remembered, accurately corresponding with what has been quoted of the 'pneumonic epidemics' of central Europe. With epidemic pneumonia, as with influenza, there is sufficient evidence in the old records that the disease affected other organs than the lungs, and concurred with a large amount of mortality where these organs were but slightly affected.

[2] Edinburgh Medical Journal, April, 1864.

Its marked character,

After speaking highly, from personal observation, of the results obtained in Germany by the 'water doctors,' the writer goes on to relate how, having been inclined to no light degree of scepticism regarding the usefulness of blood-letting in pneumonia, 'his experience of this particular epidemic impelled him, nevertheless, to the practice of it. 'Many cases,' he says, 'convinced me of its necessity in the severer cases, and these were far more numerous than I formerly would have expected.' 'It is no doubt,' he adds, 'that it may be a rare occurrence in the warmer climates of Europe to see healthy and strong people so rapidly affected by a pulmonary inflammation that death follows in two or three days; but this happened very often in our epidemic, and was the general rule where the disease was left alone. I saw several, even young, strong, and healthy people, who the day before the attack were at their work without any complaint, but who next day were nearly breathless, with all the symptoms of the most acute pleuro-pneumonia. I know a parish in the interior of our country with only 300 souls, where nineteen persons died thus affected in a very short time. Having been more than sixteen years in foreign countries, I never saw any case of pneumonia which could be compared with the common cases of this our epidemic.'[1]

and rapid fatality.

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[1] 'It would no doubt,' writes this Icelandic physician, 'have been a good lesson for the medical men of the nothing-doing treatment to see the ravages of this frightful disease amongst the helpless inhabitants in all the parishes where no medical aid was to be had, and they would

Results of bloodletting.

Coming thus quite unwillingly to the practice of phlebotomy, it is the more interesting to notice the result obtained by its means :—'The insupportable pain beneath the right nipple across the chest, the great dyspnœa which in some seemed to threaten with immediate danger, the disturbed balance in the circulation, were generally greatly relieved; and the hard, incompressible, but often irregular pulse became more regular after the loss of eight or twelve ounces of blood. The relief thus obtained was not always momentary, but would last for several hours or even one day; and although the rapid and dangerous pulmonary congestion could by no means be subdued by this remedy, it was evidently of great service to calm the storm.'

These observations from Scandinavia, referring, as they do, to the prevalence of an undoubted pneumonia, whose natural tendency was not towards recovery but towards death, are of unique value to us now as exhibiting—if I may so say—the possibilities of the disease. What has happened to the Icelanders may happen to us. In restricting our view to a particular time and country, we do less than justice to the practice of foreigners and of our predecessors, and are far too complacent in regard to our own.

Epidemic in New Brunswick.

An epidemic of pneumonia, in many respects similar to that described by Dr. Hjaltelin, occurred to the 22nd Regiment at New Brunswick. It is fully narrated by Dr.

have been able to see that their healing process of nature, which they call the natural progress, is, in a really acute and malignant occurrence of this disease, really death, and nothing but death. Nearly all the patients who were without any medical aid died in three to six days. Thus in the parish of Gardar, with about 1,200 souls, and where medical aid seldom could be had, out of twenty-four patients seventeen died. In the parish of Utskaler, with 900 souls, where nineteen fell sick, and where medical aid could not be obtained, eleven died. In the parish of Kalfatjorn, with 800 souls, nearly all the affected, this number being 14, died.' So it was everywhere, without medical interference.

Welsh in the 'Army Medical Reports' for 1867, to which valuable paper I must refer the reader.

So-called pneumonic epidemics associated with fever and dysentery.

Contrasting with such narratives we have accounts of epidemics, both ancient and modern, which, while they are described as pneumonia, differ very widely in their origin and course and response to treatment. We have spoken already of such sicknesses in the past; of the implication of the lungs, not by hepatisation, but in a general congestion tending rapidly to gangrene or purulent destruction; of the coincident liquid effusion in place of pleural lymph; of the associated dysentery or ulceration or diarrhœa. The medical history of our own day will supply similar illustrations. Thus, Dr. Bryson has preserved an account of an epidemic pleuro-pneumonia, in some ships of the Mediterranean Fleet, in 1860.[1] The disease was of the 'asthenic or typhoid type'; there was great congestion of the lungs, and in the ship that suffered most (owing, as appeared, to excessive overcrowding) scorbutic symptoms arose. Effusion into the chest was discovered in a few instances. Diarrhœa and dysentery were common. It was suspected that the sick landed at Malta communicated the affection to the patients amongst whom they were placed. There are further narratives, both at home and abroad, to a similar effect. In Ireland the mortality ascribed to pneumonia was largely increased during the years of famine, when dysentery, typhus, and scurvy were also common. Ziemssen of Berlin[2] believes that the variations of pneumonia are in proportion to the density of the population, that the mortality of typhus and of pneumonia are in close correspondence, and that the two diseases are due to similar causes.

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[1] 'Lancet,' Jan. 9, 1864.

[2] 'Edinburgh Medical Journal,' vol. iv. p. 380. Unfortunately, in dealing with large numbers and trusting to tables of mortality, the conclusions attainable are of a very general kind. From the concurrence of testimony it is made clear that *some form or other* of lung-inflammation is favoured by the causes alleged, but, except for Dr. Bryson's account, the precise condition of the organ in individual cases is not described.

The vague definition and wide associations of pneumonia in the past.

It must not be supposed from what has been said that the disease we are to discuss is chiefly seen as an epidemic. Great popular sicknesses are likely to attract the most notable and trustworthy reporters, so that in searching out the past history of many affections, the student is easily drawn in that direction. We have seen that the ancient views in reference to ordinary pneumonia were vague and unsatisfactory. In the times to which we have chiefly referred, pneumonia scarcely owned any anatomical definition whatever. Passive engorgement was always taken for inflammation, collapsed or compressed lung was never recognised as such, while pulmonary changes belonging to the mere act of death were interpreted as the signs of active disease.

Discovery of percussion and of the anatomy of the lung.

Breaking through this darkness came the discoveries of Avenbrugger and Bichat. The one proposed, though he did not perfect, a method by means of which the physical state of the chest organs should be gauged,[1] the other, many years later, demonstrated the minute structure of the lung. With the ground thus prepared and the work already begun, Laennec, pursuing the path which Avenbrugger had, in fact, pointed out, though he failed to follow it, proposed to take note of the actual condition of the chest organs by the audible report which they gave of themselves. The achievements of auscultation do not concern us here except in their

Laennec's discovery of auscultation.

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[1] Avenbrugger's work (Inventum ex percussione thoracis ut signo morbos detegendi, published in 1763) like some modern ones, met with less attention than it deserved. According to Laennec's account he would seem to have been somewhat tardy and unenthusiastic, keeping silence for seven years 'inter labores et tædia.' Corvisart revived his views 30 years later. Bichat's 'Anatomy' was published in 1801. See Laennec, Traité de l'Auscultation,' vol. i. p. 27.

Its application to pneumonia.

bearing upon pneumonia. In the enthusiasm begotten of a new discovery, Laennec sought to reach and define by its means the essential part of a disease whose boundaries and relations were as yet quite unfixed. By this new test, as he believed, an affection which had hitherto eluded description might be identified under a great variety of external forms. Pneumonia, although it could not always be seen, might by the aid of the new science always be heard.

What really distinguished commencing inflammation of the lung, then, was not the fever, nor the dyspnœa, nor the spitting, but *le râle crepitant.* This sound was always present, and although it might be imitated in other morbid conditions, yet these were most easily distinguishable by their proper signs. The opinion thus expressed was eagerly caught up and found an echo beyond the Channel. The crepitant râle, it was repeated, 'gives the earliest and surest intimation that such a disease has begun as tends to disorganisation and the inevitable loss of life, unless quickly arrested by its counteracting remedy.' [1] Thus a particular sound (whose precise mechanism is even now doubtful), was charged with deposing to the existence of a disease. The crepitant râle could give reliable information which all the other signs might conspire to negative. The instructed ear could correct the inference of the other senses.

It is in accordance with all our experience of new methods to find them pressed at first for a larger service than they can render. It required the reflection of more than one generation to perceive that the function of auscultation had here been overstrained. The crepitant râle, as it then appeared, might be heard in various physical circumstances, and was common to many conditions of lung. As such it could never of itself announce with certainty the presence of a particular disease.

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[1] Watson's 'Lectures,' vol. ii. p. 76, quoting Dr. Latham.

It was due to the circumstances of his time that Laennec was led to base the recognition of pneumonia too exclusively upon physical signs. Trusting entirely to the stethoscope he undervalued other sources of information, and failed to estimate at their true worth those more obvious signs of the disease which had been already recognised. Yet it is to him that we owe the earliest picture of pneumonia in its successive anatomical stages. Improved means of research have since made evident much that was then obscure, and enabled later observers to define the pathology of lung inflammation more strictly, but the clinical account of pneumonia, although it has since been amplified and refined upon, remains substantially as Laennec wrote it.

The labours of those who followed in Laennec's footsteps, of Andral, Grisolle, Chomel, Stokes, Williams, Addison, and others, belong to our own day, and form part of that abundant material out of which a consistent history of pneumonia has now to be constructed. The task is not without difficulty. The records of the past, as we have just now glanced at them, are perplexed and contradictory. Hardly more appears, so far, than that this affection of the lungs is of many shapes, and kindreds, and degrees of fatality. Nevertheless there runs through the ancient accounts, no less than the modern ones, two leading characters.

Two forms of pneumonia recognisable.

There is the widespread and often desolating epidemic associated with some unhealthy conditions of living, such as famine, bad lodging, and overcrowding, and bringing in its train other affections like dysentery and scurvy ; and there is the disease called by this same name, prevalent more or less according to weather and exposure, less fatal than the first, and without its accompanying diseases. In ancient times the long series of epidemics of the sixteenth century in which the whole of Europe was involved, represents the first, and certain isolated examples, such, for instance, as the pneumonia attacking the garrison of Philisbourg, the second. In our own day, Dr. Bryson's epidemic in the overcrowded 'St. Jean d'Acre,' with its diarrhœa and dysentery, or Dr. Dahl's outbreak of pneumonia in Christiania amongst overcrowded prisoners, together with many other examples variously described as 'pythogenic,' 'sewer-gas pneumonia,' and the like, represent the first, while the Icelandic epidemic in the rough winter of 1863, or that of the 22nd Regiment in the draughty barracks of New Brunswick, exemplify the second. And not only in symptoms, and associations, and mortality, is this distinction to be noted, it is traceable also in the old modes of treatment, and especially in observations both ancient and modern as to the various results of blood-letting in the two conditions respectively.

CHAPTER II.

THE FORMS OF PNEUMONIA.

Definitions of various authors—Clinical and anatomical forms recognised by these—Illustrations—Suggested classification.

Definitions of pneumonia.

A MODERN author has sought to bring diseases within the limits of terse, dogmatic description, heading his subjects with a short definition after the manner of the exact sciences. The definition of pneumonia is as follows: 'A disease expressed by severe febrile symptoms, which come on suddenly, attaining in a few hours a great intensity, and which undergo a no less sudden abatement or improvement between the fifth and tenth day.' We read subsequently of its well-marked stages, of their pathological significance, and of the sounds belonging to each. The affection is described throughout with an unusual precision; it enters upon various phases in a prescribed order, and each step in its course has its appropriate physical sign, the significance of which can be nicely appreciated. In the same spirit the history of the disease has been sketched out in a programme of parallel columns, the sounds of each period being described alongside of the supposed morbid conditions which account for them. It would thus appear that in a given case the physician would not only recognise the stage the disease had reached, but be able further to give some account of its antecedent phenomena, and to forecast with confidence something of its subsequent history.

But we are next told that, although this is pneumonia, pneumonia is not always of this form; that inflammation is wont to attack the lung insidiously and in the course of other diseases; that, so occurring, its features may be modified in various ways—so far modified, indeed, sometimes as to escape recognition altogether. It is then called (and no one can quarrel with the name) 'latent pneumonia.' One author, as angry with a disease which so escapes detection, speaks of it when it assumes this shape as 'a low, sneaking inflammation.'[1] Similarly, the writer whose definition I have just quoted alludes later on to obscure and latent forms of attack which have no resemblance to his first description, and Sir Thomas Watson appends to his graphic description of the disease the caution: 'All that I have hitherto been saying relates to acute pneumonia, as occurring in a previously healthy person; but pneumonia having that character and so occurring is a much less common disorder than most persons appear to suppose, or than I formerly thought it to be.' 'Inflammation of the pulmonary substance,' he adds, 'is apt to supervene insidiously upon various disorders which are of every-day occurrence—upon bronchitis, upon phthisis, upon disease of the heart, and upon fevers, especially the exanthematous fevers.'

In the more dogmatic enunciations of Dr. Walshe we find the varieties of pneumonia, which are indicated but not described, arranged in a table.[2] From this it appears that, in its 'secondary or intercurrent origin,' pneumonia depends on a list of eleven acute diseases—in which rheumatic fever occupies the first place, and acute diseases of the brain the last—and on a list of chronic diseases, in which pulmonary tuberculisation and cancer occupy together the first place and Bright's disease the last. We read further: 'Instead of running the ordinary course, with marked subjective symptoms, pneumonia may be completely latent. Pneumonia occurs in this form solely under circumstances of general physical debility.' The author concludes by reminding us that, in treatment, 'we must remember that the inflammatory character of the local malady is modified more or less seriously by the general state of the system.' 'It is exceedingly probable,' he adds, 'that various differences exist in the intimate constitution of many of the intercurrent pneumonias, though at present no absolute proof of the fact can be given.' Other authorities might be quoted who thus vaguely allude to declensions from the typical form of the disease. Thus Dr. Stokes speaks of 'typhoid pneumonia,' which he explains to 'include a variety of cases, seen more frequently in hospital than in private practice, in which, whether from the low state of the constitution, the complication with other local diseases, or the pulmonary affection being secondary to a general morbid condition, we find a pneumonia often more or less latent, and accompanied by extreme prostration.'[1]

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[1] See Jones and Sieveking's 'Pathological Anatomy,' p. 428.

[2] Walshe 'On Diseases of the Lungs,' art. Pneumonia, p. 375 et seq.

All this seems to be a very inadequate account of the matter. First we are told at length of a disease which is characterised by certain well-defined symptoms. It is next intimated that these symptoms undergo modifications under various circumstances, that, in fact, the modified disease is far more common than the simple one. It soon appears, moreover, that by 'modifications' no less is meant than that the disease assumes an entirely new shape. The statement amounts to this. In certain cases, in a certain definite way, the lung becomes consolidated (inflamed, as some believe), and this change is accompanied by such and such symptoms; but much more often the lung is wont to become consolidated (inflamed or not) in a different way and under different circumstances; and although all these forms of lung-consolidation alike are called pneumonia, the description given applies only to the least common.

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[1] Stokes 'On Diseases of the Chest,' p. 338.

The forms of pneumonia variously described.

That there is considerable confusion prevailing upon the subject will be further evident from comparing the views of various writers, or even of the same writer at various times. According to some the varieties or forms of pneumonia are to be associated with certain diseases or constitutional states with which it is apt to ally itself. The late Dr. Todd,[1] besides the simple disease, used to speak of pneumonia complicating gout or rheumatism, as well as of strumous, typhoid, and traumatic pneumonia. Others, again, would have us class pneumonia after its anatomical characters, as lobar and lobular, inter-alveolar and intra-alveolar, a classification, it must be observed, which is hardly possible till the death of the patient. Dr. Fuller has something of both methods. He enumerates five varieties of the disease. In the first he places cases which are especially apt to occur in rheumatic persons, and which are characterised, as he believes, by inflammation of the interlobular cellular tissue. Next comes lobular pneumonia—a form which does not occur, in this author's opinion, so often as has been supposed. The third variety includes all those cases where the disease is of secondary origin. Fourthly, latent pneumonia is mentioned. 'Its peculiarity,' we read, 'is simply that which the name implies, and which renders the mischief very likely to be overlooked.' Lastly comes chronic pneumonia—a variety which is said to be extremely rare.[2] Here, therefore, in a list where a form of pneumonia only recognisable after death occupies the first place, and lobular pneumonia —admitted to be rare—the second, the consecutive form of the disease stands third; and though this last is stated to be a very frequent cause of death, no attempt is made to sketch its clinical features, or to arrange in order the diseases most obnoxious to it.

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[1] Todd 'On Acute Diseases,' p. 368.

[2] 'On Diseases of the Lungs,' art. Pneumonia. This mode of division is obviously faulty in other respects, and must give rise to confusion from the clinical differences being mixed up with the pathological. Thus a case of pneumonia may belong clinically to the fourth variety as being latent, and anatomically to the second variety as being lobular; while

A sense of the unsatisfactory nature of such classifications is apparent in the very language of the authors, and in the changes and rearrangements to be found in successive editions of the same book. Dr. Walshe in 1871 finds it necessary to describe pneumonia no longer, as in 1844, as 'acute inflammation of the pulmonary tissue,' but less directly as 'the representative of a variety of pulmonary inflammation processes widely differing in significance, in nature, and in issue.' The acute pneumonia of the earlier edition becomes the 'acute asthenic exudative pneumonia' of the later. A writer who does not squander words needs three epithets to identify the true disease and place it beyond equivocation.

If to these accounts there be added the opinion of Dr. Hughes Bennett, that 'there are cases with all the symptoms of pneumonia yet with no inflammation of the lungs, and others with pneumonia yet with none of its symptoms,'[1] the confusion of the subject will perhaps be sufficiently exhibited.

And, turning from books to clinical records, the obscurity does not diminish. Here, for example, are two subjects whose lungs are described as exhibiting the appearances due to pneumonia. The one died by gradual sinking, with symptoms resembling continued fever, and but little embarrassment of

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pneumonia of the first variety is necessarily by its definition pneumonia of the third variety also.

[1] 'Practitioner,' vol. ii. p. 263.

respiration; the other was seized suddenly with urgent dyspnœa and acute pain, and died in a few days, suffocated.[1] Yet the disease, so far as the name indicates it, is the same in both—only 'active' in the one, and 'latent' in the other. Instances are numerous where the contrast is as striking. How far the pathologist of the present, ignorant of the histories of these cases, might succeed in classifying them in a manner which would correspond with their several clinical features, remains to be considered. The actual fact is that, in our records, no distinction is attempted. Every variety of disease would appear to be nearly associated with this common name—pneumonia. It attacks the subjects of chronic diseases of all kinds; it is a frequent attendant upon typhus fever; it occurs sometimes quite suddenly and unexpectedly as a 'complication' in acute rheumatism; while not unusually patients who have been sinking bit by bit with slow and painful lingering, die at last of pneumonia 'in its latent form.' Only rarely does it fall with fatal force upon the healthy and robust; and then its course is so rapid, and its phenomena so marked and uniform, that one wonders that so fierce and definite a disease can admit of so many modifications and varieties.

Attempts at classification.

Some years ago, when the office of Medical Registrar at St. George's Hospital offered favourable opportunity for such an enquiry, I undertook an analysis of all the fatal cases of pneumonia recorded in the hospital books during a period of twenty years. Proceeding on the ground that the clinical features of each case had claim to consideration no less than the anatomical, the problem was to ascertain first whether these examples of disease—quite unselected, and, though entered under a common name, quite dissimilar—might be arranged into natural groups, and next, supposing them to be so arranged, to ascertain further whether the clinical grouping corresponded with the pathological, so that, for instance, a case distinguished during life by certain declensions from the type of the pneumonia of the books should be known after death by corresponding declensions in anatomical respects. In other words the question to be tried was this :—Is any natural clinical classification of these cases possible, and if so, how far does such classification serve to bring together cases which are anatomically similar?

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[1] Compare, *e. g.*, Case 9 of Table IV. with Case 1 of Table V. Appendix D.

Four varieties of lung consolidation.

As the result of this labour,[1] I was led to the conclusion that all the fatal instances of so-called pneumonia occurring in this series of years fell naturally, in view of their clinical histories, into four classes. The *first* and largest class would comprise patients who died of tedious and exhausting diseases of whatever kind, such as the constant drain of an abscess, or the gradual extension of large areas of ulceration, as from bed-sores; or, generally, where lingering was unusually prolonged, and emaciation extreme. Lung-consolidation, indeed, is a familiar appearance in connection with this form of decay, and it is remarkable that the condition is described by the several narrators in terms identical with those applied to the true pneumonic consolidation, proving, in fact, that death in these cases, or in most of them, was supposed due to a 'low, insidious' form of inflammation. A *second* class would consist of the subjects of a specific fever, or of some definite affection of a secreting organ, and conspicuously of uræmic poisoning and the poison of typhus. In some of these cases the lung-affection gave rise to marked local symptoms, resembling in this respect idiopathic pneumonia, with which, indeed, it would claim, sometimes, a near alliance. In a *third* class hepatisation would seem due almost entirely to mechanical causes, such, for example, as would arise from defective power of the heart; from obstacles being opposed to the circulation owing to some valvular imperfection; from the altered constitution of the blood itself; or from any combination of these states. I shall hope to show in the sequel how the phenomena which attend some of these cases of consolidation stand related to the ordinary process of acute inflammation. *Fourthly*, hepatisation occurs in connection with that disease which the prevailing nomenclature describes as 'acute sthenic exudative pneumonia.' It is then invariably associated with pleurisy and often with pericarditis; then, and then only, it runs a definite course, involving other organs besides the lung, and, in the present day, is only exceptionally fatal.

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[1] Vide Appendix D. It was impossible out of the material before me to get more than a rough naked-eye description of the affected lung, but to avoid the inclusion of irrelevant cases I took it as essential, not only that the account of each case should describe it as one of pneumonia, but also that the lung should in every instance exhibit hepatisation, or what was taken for such. To avoid unnecessary complication, cases connected with tuberculosis and phthisis, or with secondary pyæmic deposits, were excluded from the list.

Of the four classes the last is the only one which can lay claim to distinctive features, or be classed and described among diseases as having a history of its own. Hepatisation, in the other classes, is an accident, and, while some affections are more exposed to it than others in an order that can be stated, its modes of access are so various as to admit of no general clinical description. And, moreover, although it be true that hepatisation is a feature common to all the classes, it will appear that these are distinguishable, the one from the other, anatomically as well as clinically.[1] I hope to show that in this respect each of these classes has features of its own which are perfectly distinctive, and that the more typical cases under each have little enough in common.

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[1] No doubt there are many examples of a complex kind to which it is difficult to assign an exact place. And this is what we should

Illustrative cases.

The wide field that is in fact occupied by these categories, described as a single disease, is best exhibited by actual cases. Take such an instance as the following:—A middle-aged woman, always greatly distressed by vomiting during her pregnancies, has that symptom occur in the fourth month of such a time, and with so much severity and persistence, that hardly any nourishment whatever is retained. After two months of this incessant sickness, she slowly sinks exhausted, and starved to death. Whatever conjecture is formed as to the material cause of this result, certainly pneumonia is not thought of, since there was neither cough, nor pain, nor dyspnœa, nor other symptoms that could be referred to the lungs. Yet it appears that in this woman, who thus died inch by inch, enduring the want of food for more than two months, 'the lower parts of both lungs were red, solid, and airless, from the early stage of pneumonia.' Her disease, therefore, would appear from the description to be the same with that which in six days sufficed to destroy a robust man in the prime of life. This latter, on the day of his admission to the hospital, was seized suddenly with severe rigors, acute pain in the right chest, fever, &c. This combination of symptoms led to a diagnosis of inflammation of the right lung.[1] Upon this supposition he is bled twice within a few hours, sixteen ounces of blood being taken each time. He is then ordered half a grain of tartarised antimony every three hours; and

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expect, and is in accordance with our whole experience of disease, which has always resisted being wholly comprehended in precise definitions and classifications. It is the habit, indeed, of systematic writers to describe only typical differences, leaving out of view that middle connecting ground which lies between them; but when in a purely practical spirit we descend to actual cases, these lines of demarcation, abrupt as they appear from a distance, lose their distinctness, and we find ourselves often in an undescribed middle country of which the ownership may well be disputed.

[1] Case I of Class IV. Appendix D.

the report of the third morning states that he 'has been unable to sleep, owing to vomiting caused by the antimony; he is also much purged.' So the dose is reduced to a quarter of a grain. As to nourishment, there was as little as possible of it, and that little of the most unstimulating kind. He is next blistered; and though the blister, it is said, 'rose well,' the man sank and died, violent delirium preceding death by a little, and giving place to insensibility. When this patient's body came to be examined, it was found that these energetic means had failed to subdue the inflammation, such as it was. The lowest lobe of the right lung was in various stages of red and grey hepatisation, and there was some recent pleurisy. The upper lobes of the right lung and the whole of the left lung are described as 'remarkably healthy;' nor could any disease be discovered elsewhere in the body.

Take another case different from either of these. A cabman of twenty-two,[1] who has had rheumatic fever four years back and suffered from palpitation ever since, catches a cold, and hereupon begins to spit blood. Soon his legs begin to swell, the urine becomes scanty, and he suffers from breathlessness. After three weeks of this he comes to hospital. His ten days' residence there is marked by increasing dropsy, orthopnœa, slow, irregular action of the heart, and at last delirium and death. He, too, died of pneumonia. 'The whole of the lowest lobe of the right lung was in a state of red hepatisation. At its lowest part among the inflamed tissue was a small spot of pulmonary apoplexy, while the mitral orifice was narrowed and perfectly rigid.'

Once more, a cachectic old woman,[2] who for nine months has suffered from pyrosis and vomiting, but without cough, dies by gradual sinking after six days' residence, vomiting being the only symptom recorded. Pneumonia again. 'The lower lobe of the left lung is quite solid, red, and grey, hepatisations being intermixed, and a layer of recent lymph spread over the pleura.' The post-mortem appearance, therefore, is very similar to that of the man who was so industriously bled.

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[1] Case 12 of Class III.

[2] Case 2 of Class IV.

Their various pathology.

Here, as I believe, we have illustrations of the several ways in which hepatisation is set up, examples not of the same disease exhibiting itself in different ways, but of different diseases having in common a single pathological feature. In the first case, that of the middle-aged woman, the hepatisation is the so-called peripneumonie des agonisans of Laennec, the hypostatic pneumonia of Piorry; it is not the cause of death, but the mode of it, and but one of a number of signs indicative of a gradual failing of vitality. It is misleading, therefore, to call it by a name that suggests the phenomena of a specific disease which, in this particular case, are conspicuous by their absence.

What these phenomena are, and how true pneumonia resembles in some respects a general rather than a local affection, may be seen in the marked symptoms and speedy death of the subject of the second case. The third, the cabman, is an illustration of the occurrence of hepatisation from mechanical causes. The constriction of the mitral orifice tends to keep up a constant pulmonary hyperæmia. Upon the man's taking cold, the stress of his trouble falls, as usual, upon the faulty organ, and the ultimate hepatisation of the right lung, with actual effusion of blood in one spot, which is but a further stage of the hyperæmia observed in the left, is the consequence of a direct obstacle to the pulmonary circulation. Finally, in the instance of the old woman, hepatisation, which is only one event, hardly perceived in the general failure, is again the result of obstruction; not, as before, from a strictly mechanical cause, but due to defective elimination, from advanced disease of the kidney, giving rise to a poisoned blood, whereby the already enfeebled circulation is still further embarrassed.

The associations of hyperæmia and consolidation of the lung.

How far these cases, and the groups of which they are the representatives, are, from a strictly histological point of view, to be regarded as fulfilling the definition of inflammation, may be discussed presently. I desire to maintain now that hyperæmia and hepatisation, either or both, are sometimes the consequences of pre-existing disease, and sometimes they are the expression of the disease itself. Taken alone, there is no strict clinical significance attachable to these conditions simply as such. In other phrase, there is a disease, pneumonia, of which the most prominent local manifestation is hyperæmia and hepatisation; and there is besides, the anatomical condition of hyperæmia or of hepatisation, both nearly associated with anasarca, apt to supervene in the course of many diseases, and notably connected with the many causes which tend, mechanically or otherwise, to embarrass the pulmonary circulation.

Simple pneumonia entitled to a place and name of its own.

This view of the matter, which has to be further justified in the sequel, suggests such a nomenclature as shall prevent a casual, dead resemblance being mistaken for an inherent and living likeness. Pneumonia, with all its surroundings, in its sharpness of attack, its pyrexia, its anatomical stages and associated pathology, must stand by itself. Post-mortem conditions, which resemble it in part at one or other of its stages, are not, on the assumption of a real kindred, to be allowed to take its name. The present nomenclature stands almost self-condemned. 'Acute sthenic exudative pneumonia' is quite too long a term for a disease which is at once simple and distinctive; while the various appellations of disparagement applied to its supposed associates, as 'false,' and 'low,' and 'bastard,' throw a doubt upon what is elsewhere asserted, and clearly indicate that the character of its relationship is not regarded as altogether legitimate.

I proceed to attempt the portraiture of true pneumonia, which, according to my plan, it will be no longer necessary so to characterise, since henceforth I shall cease to call anything else by its name. I shall endeavour to recall in their order the several features of the disease, and to note the connection between the general and the local signs. It will be necessary next to consider what place pneumonia should occupy in the catalogue of diseases, to fix the degree of declension from its typical pattern which may be admitted as falling still within the boundary which separates pneumonia from the numerous conditions that resemble it here or there; and, finally, as some test of the truth or falsehood of the conclusions arrived at, as well as in completion of the history of the disease, we may consider its provoking causes, the statistics of its mortality, and the results of treatment.

CHAPTER III.

NATURAL HISTORY OF PNEUMONIA.

Its aspect—Mode of respiration—Cough—Expectoration—Urinary changes—Temperature and pulse—Early prognosis.

The aspect of pneumonia.

THE physiognomy of disease, its precise look, and manner, and posture, would seem to be quite within reach of delineation and even of language. The aspect of pneumonia, indeed, is less express than some; it is not always recognisable in the features, like continued fever, or emphysema, or advanced phthisis, yet the mere look of the disease is often enough to discover it:—A flushed and somewhat dusky face, with a heavy yet anxious expression; a dorsal position, with indications of pain and restraint in breathing, which, out of mere feebleness, the patient does not seek to lessen by any change of posture. Thus, along with that special distress which arises from inability or fear to fetch a full breath, there is an aspect foreign to any mere local affection, and often nearly resembling the aspect of typhus. Yet typhus, though it assumes the same posture, renders its subject more listless and indifferent. The expression of pneumonia has more of anxiety and active distress, but it is distress which can only betray itself by the face and the obvious catching of the breath. The restless shifting from side to side which should accompany the pain, and which does accompany it when it comes alone, is here absent. The body is too weak, the perception too blunted.

Respiration quickened above the pulse.

If a patient presenting such an appearance is approached more closely, other symptoms will become evident. A hot skin, yet with less dryness and pungency than that of typhus or scarlatina; a pulse not unlike, it may be, in softness and volume to the pulse of continued fever, yet differing from it in this respect, that it is not accelerated in a like degree with the acceleration of the respirations. In fever, whether from a nervous or some more demonstrable cause, there is quickened breathing, but in pneumonia this will be more marked, not merely from the hindrance to free inspiration caused by the pain of pleurisy, but because the stress of the poison, so to speak, is directed immediately to the lungs. These organs, though as yet, it may be, without the formed products of the disease, are already disturbed in function in anticipation of the material changes which are impending.

The seizure sudden, and referred to exposure of the body.

With such an aspect and posture [1] and temperature, and with this altered ratio between the pulse and the respiration, it would hardly need more to determine the character of the disease. The earlier history of such a patient would probably tell of a sudden illness, evidenced first by shivering and sharp pain in the side, and which, breaking in upon robust health, he will probably refer, rightly or wrongly, to some external cause. He is a groom who first felt a chill when riding against the east wind, or a sailor who caught cold while working in the hold of a draughty ship, or a labouring man who on his way to church was suddenly seized with shivering on facing a blast of cold air.[2]

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[1] A patient with pneumonia sitting up in bed, or lying with the shoulders much raised, will probably have *more* than pneumonia—sometimes effusion into the pericardium, or a dilated heart, sometimes a large quantity of fluid rapidly effused into the pleural cavity.

[2] These are all actual narratives related to me by patients. I remember a man with well-marked pneumonia describing at the time the exact place on Westminster Bridge where his disease had met him; leaving the Surrey side well, he entered Middlesex with it.

I say that the symptoms just detailed, with the knowledge that the attack was sudden and recent, and marked by distinct rigor, would suffice of themselves to signalise pneumonia without having recourse to special means of investigation. It may even be said that the affection is sometimes indicated by these phenomena at a period when its physical signs are as yet absent. Instances of such a mode of invasion have been recorded by various observers from the time when pneumonia was first formularised, and when, as has been said, its diagnosis was regarded almost exclusively from the physical point of view.[1]

The evidence of cough.

As a rule, however, it is not only by the signs now mentioned that the advent of pneumonia openly declares itself. There is the cough and the material that the cough expels. Upon the first it is hardly necessary to dwell; cough is precarious and changeful, liable to be influenced by many accidental circumstances, and of itself hardly an aid in determining the diagnosis. Commonly it commences within the first twelve hours of the illness, is short and infrequent, and chiefly a source of annoyance from the deepened inspiration to which it gives rise augmenting the pleural pain. Paroxysmal cough, such as is common in bronchitis, is here quite exceptional, as is also the substernal pain which is among the earliest troubles of that disease. And while cough more or less is hardly ever absent,[1] the gravity of pneumonia cannot be measured by its character or severity. A short, ineffectual cough, early disappearing altogether (as seen sometimes in the aged) is not of good but rather of bad augury, while violent and paroxysmal cough, except with children, is almost always an indication of the bronchial mucous membrane being engaged.

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[1] We shall have to recur to this point presently. I would here refer to the kind of testimony alluded to in the text. Andral relates the case of a man of thirty-three, of full plethoric habit, who experienced the general symptoms of the disease, head-ache, general debility, numbness of the intellectual faculties, with a flushed face, injected eyes, and a frequent full pulse, at least six days before the stethoscope could detect the first appearance of the lung affection. 'All the organs,' says Andral, 'seemed to be simultaneously the seat of a strong excitement, without there being in any part real inflammation. No organ was really inflamed, but all seemed to be on the verge of it, as if they were all disposed thereto by too rich and too stimulating a blood.' 'In some cases,' says Grisolle, 'I have noticed for four days intense febrility without being able to discover on the part of any organ, and especially on the part of the lung, any lesion capable of explaining it.' In some cases, as this author thinks, though very rarely, the local inflammation would thus appear to be no more than the consequence of the antecedent fever. Andral, 'Clinique Médicale;' case 12; Grisolle, 'De la Pneumonie,' p. 187.

Of the sputum.

A more valuable sign is furnished by the expectoration. It is, indeed, in some respects the nicest and most instructive of them all, for it reveals no less than the material part of the disease in a shape so distinctive as to be rarely misleading. Sometimes, indeed, pneumonia is without this sign; children under the age of five years seldom know how to spit, and grown-up people, especially the aged, are sometimes too much enfeebled by the disease to make the effort. In such subjects the recognition of pneumonia is sensibly obscured.

Its physical characters.

The chief characters by which pneumonic expectoration is distinguished are—colour, consistency, and coalescence. The sputa, which are not abundant, are from their viscidity spat out with some difficulty, and appear in the porringer as one trembling, translucent, jelly-like mass, quite odourless, of an almost homogeneous consistence, and a colour like the rust of iron, whether the shade be deeper or lighter. Not uncommonly the little mass which accumulates in the course of six or eight hours is so viscid that the vessel containing it can be inverted without disturbance of the contents; but sometimes there mixes with the proper spitting of the disease a sufficient amount of watery bronchial secretion and of mere spittle to loose it from its hold or to prevent the separate spits from cohering into a single mass. A similar result is owing sometimes to the accident of the porringer containing water.

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[1] Grisolle noted cough at the commencement in 80 out of 90 cases. He found it occasionally absent in aged people. *Loc. cit.* p. 209 et sq.

Subject to certain variation.

A uniform colour does not always pervade the mass. Often in the course of a single day, or between day and night, the precise degree of blood colouring to which this sputum owes its distinctive character will be seen to vary. Thus at one time it will be a dark reddish brown, finely streaked here and there with blood, or even with little clots of blood speckling it, at another like amber, or again of a faint rose tint, or hardly perceptibly coloured. But what is especially to be noticed, independently of these accidents, is this—that a material whose consistence is denser than that of the proper bronchial secretion, and which is neither mere mucus nor mere pus, is intimately mixed up with the colouring matter of the blood, while the blood itself sparingly escapes.

Blood-streaked sputum, such as is seen in obstructive heart disease, is quite different from this; the viscid, vitreous, quite uncoloured sputum met with in some forms of bronchitis is also different. The pneumonic sputum is not streaked, but tinted or stained, and, although the characteristic colouring of blood may be absent for a time, although it may become greenish, or may commence as a bright saffron yellow, there is always, or almost always, some indication that blood is exuding.

First appearance of characteristic sputa.

At what period should this sputum appear, how long should it last, and what changes does it undergo with the successive stages of the disease? There is no want of statistics upon these points, but it is only the first that can be answered with any degree of precision. In the majority of cases the pneumonic sputum is distinctive as to colour and consistence with the earliest symptoms of the disease. Yet it is not the very earliest nor is its evidence always convincing. All expectoration may be delayed till the third or fourth day, or, appearing earlier, it may be at the first watery and bronchial, and only by degrees become coloured and viscid.[1] Sometimes expectoration is suppressed, or, as it were, diverted, owing, as is supposed, to the occurrence of profuse sweating or watery diarrhœa, sometimes (as in a case mentioned by Chomel) it is not suffered to appear, owing to mere ignorance of the art of spitting.

Spitting delayed or absent.

Absence of expectoration is sometimes ascribed to the particular seat of the affection. Pneumonia affecting the upper lobe has been said to exhibit this among other peculiarities. We shall have to discuss presently the reality of these. It is enough to observe now that the cases quoted in support of the particular statement have exhibited as well extreme prostration, a condition which of itself sufficiently explains the anomaly. The theories raised to account for the absence of spitting, where the apex is alone involved, upon physical grounds, have long since been disposed of.

Sputa resembling the pneumonic.

We may not say, however, that a characteristic sputum is always the accompaniment of pneumonia, nor even, without reserve, that when this kind of spitting is present pneumonia necessarily exists along with it. There is a viscid kind of nasal mucus which in some instances of inflammation of the pituitary membrane becomes uniformly tinted with blood. Passive congestion of the lungs of mechanical origin will sometimes put on a similar appearance. But the resemblance is here less close, and resides rather in colour than consistence. Such sputum is seldom tenacious and jelly-like. Or again, some accidental admixture of colouring matter other than that of the blood, or ineffectual cough, or a sensibility so blunted that cough is not provoked, may either alter the character or prevent the appearance of expectoration. We can readily recognise and account for such exceptions. But it is the rarest phenomenon in this disease to find the sputa retaining throughout a purely bronchial character. Rusty sputum in one or other of its degrees is amongst the main distinguishing features of pneumonia in the adult.

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[1] In hospital practice the details of the earliest symptoms have to be obtained from the report of the patients. In M. Grisolle's experience of 131 cases, 45, or more than a third, exhibited characteristic sputa on the first day ; 76 (considerably over a half) did so on the second day ; in six cases expectoration was delayed till the sixth day, and in one, it was as late as the eleventh. The same author observes that the anomaly of uncoloured sputum in pneumonia relates to certain epidemic constitutions, and was particularly observable in the influenza year 1837. (Grisolle, p. 217 et sq.) Chomel found the sputum white and frothy in three cases only out of 125 (Chomel, 'Pneumonie,' 183).

Microscopic appearance of the sputum,

Microscopic examination reveals, as might be supposed, numerous blood discs, and together with these, oil globules, granular cells, altered epithelium, pavement and columnar, and, sometimes, moulded fibrinous casts of the ultimate bronchules. In minute characters, therefore, there is little that is distinctive. Similar catarrhal products are to be found in the sputa of acute bronchitis. Even the moulded casts which might seem appropriate evidence of exudation (and have been so regarded by Remak and others), are as much the property of bronchial inflammation, insomuch that a special form of that disease takes its name from their presence. But while pneumonic sputum exhibits itself physically rather in its character as a hæmorrhage, it is said to be recognisable chemically from containing an excess of the fixed salts, and especially of chlorides, from the absence of alkaline phosphates, and an altered relation between its soda and potash.[1]

and chemical constitution.

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[1] In Bamberger's observations, as quoted by Dr. Wilson Fox, pneumonic sputa are said to be characterised as follows :—

Urinary changes.

Coincident with such phenomena, and supplementing them, are certain changes, more obvious than these, in the constitution of the urine. They may be divided into changes which are common to the pyrexial state, as diminution in the total volume of urine, increased density, increase of urea and uric acid; and changes which are peculiar, or, more truly, are most strikingly manifested in this particular affection, namely, diminution or total absence of the chlorides and (with a greater frequency than in any other disease not directly affecting the kidney except typhus) the presence of albumen.[1] Later on, the crisis of the affection is marked by the sudden, abundant, often excessive, return of these chlorides, in correspondence with the equally sudden disappearance of pyrexia.

Urea discharge.

The elimination of urea from day to day is subject to variations irrespective of those in the body temperature. The *total urea* during the fever (according to Mr. West's

1. They contain no alkaline phosphates, while catarrhal sputa contain 10 to 14 per cent of alkaline earths.

2. In catarrh the soda is to the potash as 31 to 26, while in pneumonia it is as ·15 to 41.

3. Sulphuric acid in catarrh is equal to 3 per cent., in pneumonia to 8 per cent. At the period of 'resolution' the chemical character of pneumonic sputa approaches the catarrhal type. (Reynold's System, 'Pneumonia,' 628.) Dr. Walshe has detected sugar in pneumonic sputa.

[1] The urine of pneumonia resembles in fact that of typhus most, and its analysis would correspond closely with that made by Dr. Parkes in a case of that disease where, with diminution of water, the urea was increased one-fifth, the uric acid was in large amount, and the chlorides entirely absent. In rheumatic fever the changes are of the same character, both urea and uric acid being increased, but to a less extent, the chlorides diminished and sometimes absent, but 'the diminution is neither so great nor so constant as in pneumonia.' The same may be said generally in reference to acute pleurisy, but it must be added that the urinary changes are here often remarkably slight, and the rule elsewhere preserved of pyrexia running parallel with excessive formation of urea 'altogether breaks down.' See Parkes 'On the Urine,' p. 258.

observations)[1] is below the normal, and markedly reduced when compared with the quantity of nitrogen ingested. The destination and disposal of this lost nitrogen, its retention in the body or elimination 'in some other form than urea,' must remain for the present matter for speculation.

Deportment of urinary chlorides.

It would appear from the enquiries of Dr. Beale that the urinary chlorides, absent, as has been said, during the acute stage of the disease, are eliminated through the lungs by means of the sputa.[2] When expectoration is very slight the channel of elimination is presumably the bowels. In such circumstances the occurrence of spontaneous diarrhœa during convalescence is thus accounted for. Again, certain exceptional cases of pneumonia where, while the urinary solids are lessened, the chlorides fail to reappear, are exceptional further in the protraction of recovery. It is supposed by Dr. Parkes that these are instances where the tissue metamorphosis is delayed, or its products are retained in the system to the damage of the individual.

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[1] 'Observations upon the Elimination of Urea.' By Samuel West, B.A., Ch. Ch. Oxon., communicated to the Medico-Chirurgical Society, November 10, 1874. The chlorides being absent, the specific gravity of the urine measures pretty accurately the amount of urea.

[2] This observation of the deportment of the chlorides was first made by Redtenbacher, who asserted, what cannot now be maintained, that the disappearance of the chlorides marked the precise period at which hepatisation took place. Neither is this so, nor do the chlorides reappear with resolution, but some days later. Dr. Lionel Beale (Med.-Chir. Trans., vol. xxxv.) has added to these observations those mentioned in the text with reference to the destination of the chlorides.

Dr. Beale points out that the method usually followed, and which was that employed by Redtenbacher, of estimating the chloride by adding a few drops of nitrate of silver solution to acidulated urine, gives a rough estimate of the chlorides both fixed and volatile; a precipitate may thus be due to an excess of chloride of ammonium when chloride of sodium is deficient or even absent. The usual clinical method, therefore, is so far fallacious, and the few cases of pneumonia where it has been assumed that the presence of a precipitate so obtained indicated the presence of chloride of sodium, do not necessarily bear that interpretation.—Vide *Med.-Chir. Trans.*, p. 327, vol. xxxv.

Appearance of albuminuria.

The frequent occurrence of albumen in the urine, and not of it alone but occasionally of blood and fibrinous casts of the uriniferous tubes, is a significant fact in the pathology of pneumonia. It is but one among many indications, to be noticed more fully in the sequel, that the disease implicates other organs than the lungs and to a degree not always commensurate with the local affection. As to the precise bearing on prognostics of albuminuria there has been some conflict of opinion. Solon,[1] who first called attention to the symptom, was certainly mistaken in connecting it with the period of convalescence. It is rather at the height of the disease that albumen makes its appearance, although from a multitude of observations it would appear impossible to associate the condition with any particular stage of engorgement or consolidation on the part of the lung. There is, indeed, considerable disparity, as Dr. Parkes points out, in the accounts of various observers of the frequency of albuminuria in pneumonia, statistics varying from nearly 45 per cent. to nothing. Metzger failed to find it once in 48 cases. Probably this as well as other features of the affection (as I shall endeavour to show hereafter) will be found to vary at different ages and seasons and in different places, so that no *general* notion is in fact attainable. In a casual observation of this particular phenomenon in its relation to the disease we are discussing, we may be led into the error of supposing that every instance of albuminous urine is to be put to the account of the then existing pneumonia, forgetting that the subjects of albuminuria due to renal disease are especially exposed to such attacks. It is, indeed, one great difficulty in acute affections and with patients seen for the first time to estimate the exact place and significance of a symptom of this kind.

There can be no doubt, I think, that the appearance of albumen in the urine is not of good, but rather of evil augury. It indicates the implication of other parts of the system in a disease which is known to become the more formidable by the secondary conditions which it induces, and especially of those organs upon which in pneumonia the task of elimination specially falls. The balance of statistics upon the subject only bears out what we should expect upon *à priori* grounds.[1]

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[1] 'De l'Albuminurie,' &c., 1838, quoted by Dr. Walshe.

Early prognosis

As the phenomena which have been thus far detailed suffice of themselves to indicate the existence of pneumonia, so, by their several characters and combinations, they may serve further to measure, in some degree, its severity, and furnish some material towards prognosis. Pyrexia is a necessary part of the disease, yet the more intense the pyrexia the graver the case; dyspnœa is to be expected, but urgent dyspnœa betokens danger. Such statements would seem superfluous in their obviousness; yet they are not even true. A temperature exceeding 103° is likely, indeed, to concur with grave symptoms, but the temperature may reach 105° at the beginning and shortly decline without the general aspect of the disease ever indicating danger. High temperature is less frequent with pneumonia than with acute rheumatism, yet the former disease may reach 107° and still the patient may recover. With a commencing temperature above the common rule of the disease there is reason to apprehend danger, which yet may never come; while a very moderate pyrexia sometimes attends, as we shall see, a particular form of pneumonia which is almost invariably fatal. Again, we get no information from the thermometer either as to the extent of

from temperature

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[1] Dr. Parkes (on the basis, however, of a very small number of cases) states the mortality of those with albuminous urine at 50 per cent., with non-albuminous urine at 14 per cent. He quotes Finger, who, out of 15 albuminous patients (the time of first appearance of albumen not being stated) lost 6, or 46 per cent.—Vide Parkes, *loc. cit.* p. 277.

lung involved or the probable duration of the pyrexia. A temperature approaching 105° on the second day may have descended to 98° before the eighth. It must be added that a falling temperature does not always coincide with general improvement, and that isolated and short-lived elevations of temperature, often unexplained, are not uncommon, and a frequent source of misjudgment.[1] Hence, what is true of all the symptoms of pneumonia is true especially of its temperature, that an isolated symptom can never be trusted to measure the gravity of the affection.

and pulse.

Much greater reliance is to be placed on the pulse. A pulse which *keeps* over 120 in the adult male, whatever its character, must always cause anxiety. It does so the more, as I think, when it approaches in character the pulse of continued fever in being soft and compressible. A dicrotic pulse, however, occurs often as a temporary condition, and its indication is uncertain. Of the significance of the respiration rate and pulse-respiration ratio it will be better to speak presently. Many circumstances may influence these phenomena, and it is in the progress of the disease, and not at the first encounter, that we learn to appreciate them.

And indeed with the affection as yet but half investigated, I know not how much of error and misconception may attend the endeavour to anticipate its final issue. A prognosis of any disease at first sight is but an idle guess at the future. In the case of pneumonia, if we may not withhold an opinion which must be based upon very insufficient grounds, I think we shall form it best from observing how on the whole the patient bears his suffering. The *impression* (so to call it) which the disease makes, as indicated by his aspect and gestures, and the degree of change that has come over him mentally and bodily, will measure the gravity of the peril which awaits him better than either thermometer or sphygmograph.

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[1] See Appendix B, Temperature, and the Illustrative Cases, Appendix C.

Wunderlich would call all cases severe where the temperature records 104°, the pulse more than 120, and the respirations more than 40 during the height of the disease. The rule, or any similar one, might apply usefully no doubt in gathering and sorting statistics; yet until statistics *are* so arranged, and we have learnt therefrom the actual mortal rate of the disease in these circumstances—'a task which,' Dr. Aitken says, 'will hardly be accomplished in our days'—the term 'severe' is quite arbitrary.

CHAPTER IV.

THE NATURAL HISTORY—*continued.*

Physical signs of the disease—Their time of occurrence and duration —The progress of the patient towards recovery—or towards death.

Of the physical changes within the lung,

Thus far I have designedly delayed the consideration of those elements in pneumonia upon which it is too much the custom to concentrate attention, and which concern the physical condition of the lung itself. Yet with such a patient as has just been described it would be obvious, even to an unpractised observer, that the breathing apparatus was directly implicated. And, in fact, the lung, or some portion of it, is at this period—or, by possibility, is only about to become[1]—the seat of a morbid process which, by its successive changes, serves to measure the stages and determine the duration of the disease.

This process commences with hyperæmia and is completed with the exudation of fibrin and escape of blood into the pulmonary air sacs. These two events, first an effusion of serum into the vesicular structure of the lung; next, the passage of a coagulable plasma which moulds itself in its new residence so as to consolidate the heretofore spongy texture of the organ, are common to all cases of pneumonia.

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[1] Wunderlich says, 'During the first appearance of fever there is often no other symptom. It is very seldom that any auscultatory symptoms can be found in the thorax.'—*Thermometry*, p. 374.

Yet they are not two, but rather stages of a single process. An exudation, at first watery, is ever becoming more and more coagulable as more and more of the material of the blood exudes through the vascular walls, while, in places, by little ruptures, the blood itself pours out. It is thus both an exudation and a hæmorrhage. The rate of its progress and variable duration of its several stages constitute whatever modifications the disease legitimately admits of.

and corresponding physical signs.

Now the auscultatory signs, the sounds that meet the ear from day to day as the disease progresses, are in accordance with these events. At first, with the earliest effusion of watery serum into the vesicular structure of the lung, there arises, as the inspired air reaches to the ultimate bronchules—at the end, that is, of each long-drawn inspiration—a *minute crackle*, not loud but distinct, breaking in abruptly upon the soft sighing of inspiration.

Minute crepitation.

It is a poor thing to attempt the description of sounds by words. No more can be done than by likening them to other sounds which are more familiar; even then the comparison does not seem equally apt to all hearers, and is of doubtful assistance to any. Nevertheless the minute crepitation of pneumonia may be very closely imitated. A piece of quite dry tissue paper pressed up into a ball and squeezed in the hand with a varied amount of force, so regulated as to give by practice a finer and finer crackling sound, will at last represent exactly *the character* of fine crepitation. Alternate pressure and relaxation by the hand, in time with the time of respiration, will even convey something of the general effect that the ear receives when listening at the chest itself, although in *fineness* it hardly comes up to the minute bronchule crepitus.

Again, minute crepitation is imitated to perfection, as Dr. Williams first pointed out, by the sound which is produced by rubbing one's own hair between the fingers close to the ear, although the illustration has the single drawback that in mechanism it is quite dissimilar to the pulmonary crepitus.

Its special characters.

It is necessary to refine somewhat in speaking of this particular sound, because mere crepitation, that coarser crackling which will be got out of thin dry paper by crumpling it up roughly in the palm, is of different significance and probably different mechanism. We shall have to speak of this latter in connection with the resolution stage of pneumonia. It is elicited by means of a forced inspiration, and indicates probably the reopening by the violence of the in-current of collapsed but empty bronchules. It is in accordance with this view of its origin to find this coarser or larger crepitus disappear for the while, so soon as by a full expansion of the lungs the bronchules have been once expanded.

It is different with the crepitus we are now concerned with, and which depends, as some conjecture, upon the passage of air through fluid in the smallest bronchules. This not only abides under all circumstances, but, in most cases, its time and place are unvarying. It will occupy, I mean, not the whole inspiration, but only the latter end of it. A single inspiration may, indeed, sometimes be obtained without it, by purposely shallow breathing on the part of the patient. The in-current of a shallow breath does not reach the spot where the crepitus is produced. But the needs of respiration are such that (except as I believe where very acute pleuritic pain is present) a suspension of the sound from this cause will never occur naturally.[1]

Of the first stage of pneumonia this is the earliest and sometimes the single physical sign. Whether or not some perceptible lessening of the natural resonance arises along with it is a refinement it is not worth time to dwell upon.[1] Theoretically one supposes that it would be so. It is more to the purpose to bear in mind that any marked degree of dulness at this period (while as yet the small crepitus is unaccompanied by blowing respiration) will be probably due either to the interposition of thickened pleura or to effusion into the pleural cavity.

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[1] See Appendix A: the crepitant râle.

Not necessarily indicative of pneumonia.

While this minute crepitus is necessarily associated in the mind with pneumonia, since it occurs amongst its earliest physical signs, pneumonia may exist without it, and it may exist without pneumonia. The absolute probative value of this sign has in fact been much exaggerated. It proves no more than that the ultimate bronchules contain a superabundant amount of fluid of whatever origin. Hence, in so far as it is a sign-proper at all, it is a sign-proper not so much of pneumonia as of capillary bronchitis, or of the œdema which is preliminary to pneumonia. Regarding the latter disease anatomically as an exudation of plastic material into the air cells, minute crepitus is not its sign but its immediate precursor, a signal that pneumonia is to be expected, not an evidence that it has begun.

In such a case as we have before us, however, where there is pyrexia of a certain sort, and 'stitch,' and rust-coloured sputum, the meaning of minute crepitation is not doubtful. Nor does it long exist alone. Often, indeed, so soon as the crepitation is heard, there is heard along with it an altered character of breathing—it becomes blowing, sniffling, tubular, or even markedly metallic—while, at the

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[1] Skoda maintains that the percussion sound remains unaltered (Markham's translation, p. 289). Dr. Walshe believes, on the contrary, that it 'acquires to a slight extent the characters of Type I., the amount of tone diminishing, the pitch rising.' He adds, 'Any serious change in these respects renders it possible that an intermingling of actual exudation has already occurred.'— Walshe 'On the Lungs,' p. 350.

Physical signs of hepatisation.

same time, the percussion note elicits total absence of lung resonance, and the hand laid upon the place discovers exaggeration of the natural vocal thrill as the patient speaks. Such signs are the direct and sufficient evidence of solidification of lung by the filling up of its vesicular portion. The larger tubes only remain patent, while the increased density of the parenchyma surrounding them modifies (in many ways, owning many names) the sound of the entering and retiring air, so as to liken it to the blowing of wind through a pipe.[1]

By this consolidation not only is the natural vibration of the lung with the act of speaking increased, but the voice of the speaker is conveyed to the listener's ear with a ringing clearness and intensity which will be more or less striking according to its natural tone. The phenomenon becomes at once apparent on comparing the solid lung with its fellow, and is most distinct with a bass voice. We are not to expect it in the high-pitched notes of women and children, while it is to be remembered that both the voice and its vibration—both *vocal resonance* and *vocal fremitus*—may be deadened or lost by the interposition of a quite thin layer of semi-fluid lymph. The voice in that case, consolidation notwithstanding, instead of striking the ear from its shrillness, may sound distant and quavering.

Occasional abnormalities.

By keeping in mind the physical condition on which these signs depend, the observer is prepared for their occasional absence and various modifications. There are certain phenomena, however, in the acoustics of this stage which, while they are sufficiently frequent to need mention, are still awaiting explanation. It will happen, for instance (and in cases not otherwise exceptional), that the solid lung, instead of yielding to percussion the usual dull wooden note, gives a resonance which suggests the neighbourhood of an empty cavity, deadness of sound, that is, along with tympanitic ring, or else that ringing which has been well called 'metallic.' The impression thus conveyed by percussion may be still further confirmed by auscultation. Cavernous respiration will often concur with this metallic note, while the conclusions to which such signs point may be yet further strengthened by the existence of very perfect pectoriloquy. Whatever may be the true explanation of these signs,[1] it is quite certain that they may be met with in lung that is simply consolidated. In the stage of pneumonia with which we are at present concerned, such abnormal sounds would not be really misleading. True pneumonia breaking up thus early, so as to produce cavity, is at least a very rare occurrence; it is also one which would be supplemented by a group of symptoms themselves not likely to be overlooked. There may be other anomalies,[2] not in the sounds themselves, but in the order of their occurrence. Not seldom, for instance, it will happen that minute crepitation is not heard at all at any period. The preliminary stage of the

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[1] I believe it to be true that the lung consolidated by means of a solid deposit occupying its air sacs, yields a variety of tubular breathing which is peculiar and distinctive, but I demur to the conclusion that this is tantamount to saying that pneumonic consolidation may sometimes be recognised as such by the ear alone.

Time of occurrence of hepatisation.

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[1] See Walshe 'On the Lungs,' p. 352. See also the same author, under 'Percussion' and 'Auscultation,' upon the variations in character of bronchial and tubular breathing and their probable significance.

[2] A not infrequent source of error arises from the presence of pleural effusion along with pneumonic consolidation. The lower lobe of one lung solidifies, and at the same time fluid is poured into the pleural cavity. Now the solid portion of lung being incapable of compression, the action of this accumulating fluid is to displace it, pushing it up against the superior and unaffected lobe, which thus becomes compressed and in time carnified. Although such a condition is quite recognisable, both before and after death, yet it often doubly misleads. The displaced yet solid lung is, during life, taken by its sounds for a lung compressed merely, and the carnified upper lobe, *post mortem*, is taken for lung in an earlier stage of inflammation than the lower.

systematic writers is thus apparently stepped over. With the commencing symptoms of the disease, the lung, or a portion of it, is found to be solid; the signs of hepatisation not being preceded, so far as is known, by the signs of engorgement. Such a mode of origin by no means invalidates the diagnosis of pneumonia.

By dividing the disease too strictly into distinct parts, each with its own period and proper signs, we do in fact take the life out of it. The stages which are described as successive are often concurrent. At a given point in the course of the affection the implicated portion of lung will often be found to exhibit, not that particular morbid change which, according to books, should correspond with its date, but every phase of change from hyperæmia to grey infiltration. At the same time it must be admitted that hepatisation at the commencement of pneumonia is exceptional; it is especially so if complete, if there be tubular respiration *only*, without crepitant râle. The physical signs of consolidation will suggest, though they do not prove, that the affection has lasted for more than twelve hours.[1]

Progress of patient.

Let us return now to our patient, and note what further symptoms mark the progress of a complaint whose advent is thus unmistakably declared. At an early period in the career of his disease there comes an interval, not, indeed, of pause (for the fever remains high, the urea discharge in excess or defect, and both it and the respiration subject to oscillations), but of obscurity, when the course of the disease, exposed as it is to accidental and temporary exacerbations, can only be faithfully ascertained by means of auscultation. For the changes which are now taking place within the lung, changes which are a part of the natural history of the disease, do not announce themselves visibly or by obvious signs. Thus the passage from engorgement to consolidation, the spread of the disease in local area, even the implication of the fellow lung, are events which the ear alone can detect.

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[1] Here are some statistics upon this subject:—

Grisolle says, 'I have established the existence of bronchial blowing hardly 12 hours from the commencement of the pneumonia. With two-thirds of the patients crepitation is mixed with tubular blowing from the third day.' (Grisolle, p. 295.) Dr. W. Fox says, 'Bronchial breathing may not appear till the second or even the fourth day, and this appears more commonly the case with pneumonia of the apex.' He mentions 12, 24, 48 hours as possible times. Walshe does not allude to the point, nor does Chomel. Some cases of rapid hepatisation will be referred to subsequently.

Now, too, comes the time of suspense. While these pulmonary phenomena are still in progress, the variations of body-temperature, the rate and ratio of the pulse and respiration, and, above all, the face and manner of the patient, must be carefully scrutinised. From any of these quarters the first signal may come either that the stress of the storm has passed, or that it is about to overwhelm its victim. In the majority of cases, between the third and the eighth day from the initial rigor, a sudden lull comes. The temperature of the body drops by one or two degrees, or more, and the pulse falls along with it, insomuch that sometimes in the course of forty-eight hours both may be below the standard of health. At the same time, and as suddenly, the febrile aspect is lost, and the habitual expression returns.

The crisis.

Up to the very eve of this event (which occurs indifferently—at any time of the day or night), the temperature may have been steadily rising, or it may have maintained an equable, or nearly equable, rate throughout for like periods of successive days. Upon the occurrence of crisis the fall is sometimes so considerable as of itself to threaten danger. Ziemssen has reported a fall of ten degrees in sixty hours. The pulse usually becomes lessened in frequency at the same time with the lessened heat; but it does not fall so often as does the temperature, below the normal rate, and will sometimes vary in frequency from day to day with no corresponding variation of the thermometer. The diminution of respiration at this time is the least constant and the least reliable [1] of all the phenomena of crisis.

Urinary changes.

Concurring with these changes lithatic deposits are often noticed in the urine, and the amount of urea discharge may diminish. It is important to observe, however, that these signs coincide with the stage of 'resolution,' an event which, so far as physical signs inform us, is not coincident with crisis, but posterior to it.

Crisis not peculiar to pneumonia.

This phenomenon of crisis, I say, is not constant with pneumonia, neither is it confined to this disease. In most of the so-called specific fevers signs of improvement may be equally sudden, the fever quitting its hold as in a moment, whilst profuse perspiration, or diarrhœa, or the discharge of highly lithatic urine supervening, come to be regarded in the light of 'critical phenomena.' Only in pneumonia the sudden disappearance of pyrexia is so marked and frequent as to be reckoned in its ordinary history.

Critical sweating or diarrhœa.

Symptoms supposed to be 'critical,' like diarrhœa and sweating, may in fact be mere accidents of the time. The effects of drug treatment may simulate them. No doubt in this affection, as in others, a critical change is sometimes announced by sweating; yet sweating may occur in pneumonia quite apart from crisis, and of itself is neither of good nor evil augury. Similarly diarrhœa, arising, so to speak, spontaneously, seems sometimes a part of the process by means of which the individual suddenly reverts to health; but this same symptom is not uncommon as an accident of the disease, and in no obvious connection with any particular stage of it.

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[1] See Appendices B and C upon these points. In reference to the comparative changes in pulse and respiration at this period, Dr. Walshe mentions that the fall of the latter is often the greater, insomuch that the perverted pulse respiration ratio upon which he so much insists may wholly disappear. I have sometimes observed the contrary. The crisis may be succeeded (as it may be in fever) by abnormally slow pulse, while respiration, less frequent indeed than before, owing to the departure of pyrexia, is yet frequent, owing to the presence in the lung of the material products of the disease, and the pulse respiration ratio is more perverted than ever.

We might expect that a change in the character of the sputa would be among the earliest signs of the termination of pneumonia. It was so believed of old. Boerhaave and Cullen and Huxham only followed Hippocrates in supposing expectoration to be the natural mode of exit of the disease, and attempting to promote it by their drugs. Later on, the belief in crisis by expectoration had the support of the elder Frank. Yet in truth the character of the sputa gives no more than a tardy indication of the local progress of the disorder. The crisis, as we shall see presently, often antedates the earliest physical signs of amendment on the part of the lung, and may be wholly completed while the sputa are still unchanged.

Particular days for crisis.

I have said that this disappearance of fever in pneumonia may be looked for between the third and eighth day from the initial rigor. Crisis does not always fall within these days, but the collection of a large body of cases shows that the great majority may be so included. Dr. Wilson Fox, in his valuable essay, has collected the statistics of several observers on this point. His tables show that in 374 cases as many as 324 had that issue between the third and the eighth day, the seventh and the fifth being the favourite days.[1] Crisis on the ninth day is uncommon, and, longer delayed, it is hardly to be looked for at all. A sudden amelioration may indeed yet come, but its phenomena will not be analogous with those we are now considering, while in other respects such cases will be seen to differ from the ordinary pattern of pneumonia.

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[1] Reynolds's 'System of Medicine.' vol. ii. p. 650. The numbers are from the 3rd to the 8th day, both inclusive, 31, 33, 78, 50, 96, 36. The absence of crisis, or, in the language of the learned, 'defervescence by lysis,' would be accounted for sometimes by the persistence of serous inflammation, which, originating with the pneumonia, outlasted it.

Delayed, or irregular, or false crisis.

No doubt crisis may be absent altogether in some instances; yet more often it only passes unobserved, because the body temperature fails to indicate it, or indicates it uncertainly. Thus, the thermometer may begin gradually to descend towards the end of the second week; or it may fall and rise irregularly without ascertainable cause, or apparent detriment to the patient; or a lowered temperature may occur suddenly in such time and manner as to resemble crisis, yet presently depart, the pyrexial temperature returning or an uneven course of temperature succeeding. From observations of this kind, and the fact that these irregular temperature changes receive no explanation from the condition of the patient, and are in fact no true index of it, the first asserted value of the thermometer as applied to pneumonia is hardly maintained.[1]

The doctrine of critical days[2] taught by Hippocrates, and concurred in by Andral, has been lately revived by Traube. The discussion as to whether crisis favours the uneven days or falls indifferently upon odd and even, seems to belong properly to a past age in medicine, and I shall not stop to consider it. On the other hand, there are two observations bearing upon the pathology of the disease (to be further noticed in their place) which cannot fail to arrest attention. One is, that the crisis of pneumonia often precedes[3] the earliest physical sign of resolution, considered as a mate-

Relation of crisis to the local affection.

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[1] See Appendix B, 'Temperature.'

[2] See Grisolle, 315.

[3] In 72 out of 192 cases cited by Grisolle, a notable diminution in the febrile phenomena preceded by one or several days the stethoscopic changes; in 92 it was simultaneous with these, in 26 the stethoscopic signs of amelioration were the first. Thus crisis, so far as was seen, was non-simultaneous with the material lung changes in 98 cases—more

rial lung process. The other is, that the time and mode of crisis are not influenced by the extent or site of the local affection.

Is it analogous to the defervescence of variola?

An analogy has been supposed to exist between pneumonic crisis and that sudden defervescence which marks the completion of the eruption in small-pox. The comparison is used in testimony of the unity or parallelism existing among diseases, and is made to serve for an instance of morbid elimination according to some supposed law of nature which stands in need of illustration. As a representation of clinical experience it cannot be accepted. Crisis, as we have just seen, is not a constant phenomenon, it does not mark the completion of any morbid process that we know of, nor stand in a constant relation to any. Just as the fever of pneumonia may precede all local signs of it, so in its duration it may go beyond or fall short of them.

The sequel of hepatisation.

Here, then, the history of pneumonia, so far as it can be generally described, comes to an end, for from this point its phenomena will vary according as the issue tends towards recovery or towards death. Yet in either event, with the consolidation of lung and final expulsion of air we lose count for a time of the successive steps in the progress of the exudation. For the intimate changes which that product is destined to undergo (and which remain to be described) are not such as to modify at once the acoustic signs. From solid to semi-fluid, from a hepatised or liver-like lung to one that has become soft and yielding, are indeed transitions which the ear may mark, but, between these extremes, there are many finer gradations of change which imply but little alteration in the gross density of the lung

than half. Some critical phenomena or other coincided with resolution in 34 out of 130 cases. See Grisolle, pp. 300 and 311.

and own no separate expression in auscultation. The precise physical condition of the lung, therefore, at this important stage of the affection is mere matter for conjecture. The organ still announces itself to the ear as solid and airless, while a persisting pyrexia may arise from accidental causes and concur with salutary changes in the exudation, or else be among the signs of purulent metamorphosis and inevitable destruction.

Physical signs of commencing resolution:—

In the case of recovery, what is that exact degree of change in the consistence of the exudation which suffices to produce the earliest modification of the respiratory sound? The question can hardly be decided upon direct clinical evidence. Certain it is that the most constant and intelligible, and often the earliest intimation of commencing resolution, consists in altered quality—lessened intensity, as we say—of the tubular breathing. Successive modifications of this respiration may be traced sometimes through all gradations, from a metallic blowing as through an inflexible tube, to a respiration which is harsh merely, and wanting in the soft character of the healthy vesicular murmur. Sometimes these transitions will be more abrupt, and sometimes their gradual character may escape observation, for it must be allowed that the phenomena of resolution, coinciding as they do with the safety of the patient, are followed with a less lively interest than those which concur with the active stages of the disease.

Lessened tubularity.

Redux crepitation

Along with this lessened tubularity, but more variably than it, occurring so capriciously, indeed, as to be of little practical value, is the sound due to reopening tubes with the unfolding of disused portions of lung. This harsh creaking (the 'crepitatio redux' of systematic writers) which indicates probably the unsticking of alveolar passages or of the bronchules leading to them, is accompanied sometimes by some coarse bubbling. The crepitation, insignificant as it is in importance, coarse as it usually is in character, is yet sometimes, as has been intimated, mistaken for the minute crepitation of the commencing disease, which, accordingly, is believed to have started afresh. Under that error cases are sometimes treated, and even placed on record, as relapsed pneumonia.

mistaken for crepitant rhonchus.

But though the physical signs of resolution are apt to be heard in this order, the tubular breathing becoming less tubular before either rhonchus or redux crepitus makes itself heard, it would not be true to say that the process of recovery fulfils in an inverse order the process of the advancing disease. In the oncoming stages the most perfect tubular breathing may be announced quite suddenly ; in resolution its departure is never sudden. Again, the crepitation of resolution is quite uncertain in time, duration, and place of occurrence. Weeks after the patient has recovered it may be heard from time to time in deep inspiration, or it may never be heard at all from first to last.

Diminishing percussion dulness.

Following the other signs, and like them variable as to time, is a lessening in the brassy ring of the voice and a percussion note no longer absolutely dull. It is not uncommon, however, for dulness of some degree to remain for an indefinite time when the breathing is restored to its normal character, a condition which probably depends upon a thickened pleura.

Return of urinary chlorides.

Corresponding pretty closely with the phenomena of resolution is the return of the urine to its natural constitution, the reappearance—sometimes in excess—of the chlorides, urea discharge[1] in normal quantity, and absence of albumen, although the continuance of this last is very uncertain and anomalous.

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[1] According to Mr. West's observations, already quoted, the total urea in convalescence is at first reduced and subsequently rises to that of health.

Convalescence

The convalescence of pneumonia is brief and secure, and recovery usually complete. Considering the acuteness of the symptoms and their wide range, and the fact that chronic lung disease generally entails uncommon wasting, it is subject for remark that the loss of flesh should be so little. There lingers no after-effect and no acquired susceptibility, nor does the past illness render the individual more liable to future attacks than his neighbours.[1]

Duration.

The duration of the affection from its first rigor to complete recovery will vary with the subject, with the treatment, and with the keenness of the competition to present favourable results. We have seen that the acme of the disease is to be expected within eight days, but complete recovery may be delayed by the extent of lung involved, by the amount of pleural effusion, by bronchitis, by the feeble recuperative power of the individual, by the state of the weather, and many other things. It is enough to say, therefore, that lingering convalescence is rare, and that in the majority of pneumonic cases of ordinary severity the patient is well in the third week.[2]

The modes of fatal pneumonia.

But there is another side to the picture. Pneumonia may destroy life in several ways. Either the material thrown upon the lungs interferes to a fatal degree with the function of respiration, or the process of inflammation assumes a destructive character so as to spoil an organ essential to life, or, thirdly, the demands which the disease makes upon the vitality of the individual are excessive and he dies exhausted. Of the tendency of the affection in any of these directions, we have, as I have said, little means of judging beforehand. That pneumonia is grave in proportion to its extent and most fatal to those who are already enfeebled, though it may seem like a truism, is not necessarily true. Yet of course the fear of death from apnœa,—of 'suffocative pneumonia,'—is heightened by the knowledge that a large area of lung is involved. In that period of suspense of which I have spoken, where auscultation ceases for a while to be of service, the general condition of the patient is not uninstructive. Even before the time for crisis has arrived, urgent dyspnœa, which forbids natural sleep and is made worse by the sleep procured by narcotics; expectoration scanty, viscid, and bloody, which clings to the lips and teeth; muttering of irrelevant matters; a flushed, just dusky, complexion, and above all an expression and perception hour by hour becoming obviously duller, these are evil symptoms apt to occur together; they are not necessarily fatal, nor so formidable as those which for distinctness are called 'typhoid,' yet, with whatever temperature and whatever pulse, they are full of peril. To such a patient any hour may bring the death summons, nay, at any moment almost, with no typhoid symptoms at all intervening, we may get notice, from the breath becoming panting and the face blue, that the heart is giving in and life as good as surrendered.

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[1] It should be added that recovery is sometimes delayed by the persistence of pleurisy and its attending pain on deep inspiration. Also that the observation of the small wasting that pneumonia entails does not always hold. Exceptionally there is marked wasting with a short attack and where there is no suspicion of phthisis or tubercle.

[2] See Illustrative Cases, Appendix C.

Death by apnœa.

In such circumstances our knowledge of the actual physical condition of the lung is far from accurate. As a matter of fact and statistics it is usual, upon the occurrence of death, to find it grey more than red, and not seldom so soft and disorganised that one cannot but wonder at the absence of distinctive auscultatory signs. Even when life is prolonged and death heralded by pinched face, drying tongue, sordes about the lips and teeth, and such lingering as makes the final sinking seem rather the result of exhaustion than of apnœa, it is seldom that the actual fact of breaking down of lung tissue is demonstrable by the stethoscope.[1]

Occurrence of general pulmonary œdema.

Yet often near the end—it can hardly be said in anticipation of other signs whose meaning is not doubtful—there may be added to the physical indications of consolidation those of general pulmonary œdema. The sub-crepitant râle heard at one or both bases, or universally, is a sound of ill omen at this period, as signifying the access of general congestion, a common preliminary of total failure.[2]

Stealthy advance of purulent conversion.

We are not much aided in detecting purulent conversion[3] by any special change in the course of the general symptoms. Contrary to what might have been supposed, the breaking up of the lung is unaccompanied by that particular event which elsewhere is apt to mark the progress of purulent change—I mean rigor. Not only so, but in many instances of rapidly fatal pneumonia, where almost the whole lung becomes laden with pus, the symptom of shivering has been wanting from first to last. Such cases belong, indeed, to a class of their own, and must be studied separately.

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[1] One reason for this no doubt is that, while these changes are rapid, there is a proper unwillingness needlessly to meddle with those who are obviously dying and past help. There is, indeed, a form of pneumonia (if it can claim that name) where the affected lung will so break down at a number of separate points or centres that auscultation is able to discover in the more advanced of these the existence of actual excavations, and the physical condition is thus accurately known before death.

[2] And it happens sometimes, to mislead the unwary, that without obvious amendment or obvious decline the auscultatory signs will alter in this way. Over a portion of the solid lung there will be heard, besides tubular respiration, a bubbling crepitation, coarser and, if I may so express it, moister than the minute crepitus, while it is quite unlike the gushing noise of redux crepitation. There is in fact nothing distinctive about this sound; it may be heard with humid bronchitis and heard at the commencing stage of hepatisation; it needs no more for its production than excess of watery secretion in the medium-sized tubes. All that need be remembered concerning it is its unimportance.

[3] It is common to speak of 'purulent conversion.' How far the term is histologically accurate in implying that the pus corpuscle is absent from the earlier stages of the pneumonic process we shall have to consider presently.

Indications furnished by the pulse

Does the course of temperature or the pulse afford a more certain indication of the fatal course of the disease? Not, I think, at the very time, or by signs that can be surely interpreted. The thermometer alone may even mislead, and, exceptionally, will record a falling temperature when the patient's aspect betokens rapid decline. The pulse is more reliable. For although, as has been mentioned, cases of great gravity, ultimately fatal, are occasionally remarkable for slowness of pulse, yet it is the rule nevertheless that the frequency of the pulse measures the severity of the disease, so that where this is above 120 the other symptoms are usually grave. Sometimes the pulse, with the full, soft character which, in these days, we get to associate with the disease, becomes distinctly dicrotous for a while without our being able to connect that phenomenon with any special change for better or worse in the patient's general condition. A pulse which is small and thread-like, though of no great frequency, may coincide, as I have seen, with very grave symptoms.[1]

Yet the index afforded by the pulse is sometimes misunderstood. A feeble, or even intermitting, pulse at the wrist may be wrongly attributed to want of power on the part of the left ventricle, and the inference may be thence drawn of general muscular failure. But the character of pulse produced by a weakly contracting ventricle may be equally due to the fact that, owing to pulmonary stasis, the blood delivered to the heart in the short interval of diastole is itself in defect. The ventricle contracts with vigour, but there is little for it to propel, and the small and weak pulse is the result, not of imperfect propulsion, but of insufficient blood. It is sometimes made evident that such is the case from the pulse quickly regaining its old character, and the face at once recovering colour, so soon as the physical condition begins to mend.

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[1] Regarding the sphygmographic tracing of the pulse in pneumonia I have nothing of my own to offer, nor do I know that any special value is claimed for such records in the case of this particular disease. Dr. Galabin (in Guy's 'Hospital Reports,' vol. xix.) states that pneumonia in its effect upon the pulse is 'intermediate between surgical fever, erysipelas, and acute rheumatism on the one hand, and typhus and typhoid fever on the other; it is dicrotic, but less so in proportion to the temperature than in typhoid; it also, in the earlier stages, bears a higher pressure, the points of the curve are sharper, with upstroke more nearly vertical. Of the two causes of dicrotism, lowered tension and suddenness of heart action, the latter, more than in typhoid, approximates to the sthenic dicrotic pulse.'—Vide *loc. cit.* pp. 79, 80. See also Dr. Wilson Fox, 'On the Tracings of the Pulse in Pneumonia,' 'Reynolds's System,' vol. iii. p. 737.

and tongue,

At this time the tongue is a valuable guide. When this becomes dry and baked, as it sometimes does, though the temperature should not be extreme or rising, nor the pulse up to 120, the patient's state is yet to be considered critical. The tongue at the commencement may be red and slimy, or thickly furred, or natural, just as it happens; there is little to learn from it; but when the fever has endured for a while it becomes as nice an index of the general condition as in the specific fevers.

and by the sputa.

As regards the sputum it is often absent at this stage. It was an observation of the old physicians, which modern experience confirms, that liquorice-juice or prune-juice expectoration is of bad augury in pneumonia. Yet in regard to the diagnostic value of the symptoms it must be added that while, in most cases, it does but concur with a group of others all tending to the same conclusion, in some it may accompany the early stage of hepatisation, and even simple engorgement. Trousseau speaks of the diffluent character of the sputa as being more certainly of evil augury.[1]

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[1] See also Andral, 'Clin. Medicale,' i. 95.

Purulent infiltration rapid in progress.

The possible duration of grey infiltration, the period, that is, for which a patient may survive after that change has been accomplished, as well as the precise stage of the disease beyond which recovery is impossible, these are points to which, obviously, from the ignorance which has just been confessed, it is impossible to reply definitely. And even if we could fix exactly, by some notable phenomena, the very commencement of each anatomical stage in the progress of pneumonia, still the age and strength of the patient, and, more than all, what I must call the *particular type* of the disease, would so modify the result as to render impossible any but the most general statements. Certainly it is rare to find the patient linger long enough for his complaint to assume the general aspect of chronic illness. The activity of fatal pneumonia remains to the last, and although the several steps in the decline may not be appreciable, yet the disease seldom makes pause. Only in exceptional cases is death so long delayed as to seem due at last less to the affection itself than to the condition in which the patient has been left by its agency.

The cardinal points in the disease

In this sketch of the main features of pneumonia, I design no more than to place before the reader the cardinal points of the disease. Certain details remain to be filled in; certain positions, perhaps, to be defended. But the general character of pneumonia is not matter for doubt or controversy. It is both a local inflammation and a special form of fever. As an inflammation it is remarkable for the distinct stages it undergoes, for the deportment of its histological elements, for the preservation of structural integrity amid changes which in other circumstances imply permanent damage. As an affection of the general organism it is no less remarkable for a sudden access of pyrexia, whose character and duration are not measured by the lung mischief, and whose time of departure, occurring within a set time, irrespective of recovery on the part of the lung, is equally sudden and characteristic. If the pyrexia is symptomatic, why is it not a truer index of the inflammation that provokes it? If, on the other hand, the lung affection is no more than a symptom of a specific fever, whence does this fever arise; how, with such unexampled suddenness, should mere exposure suffice to produce it in a healthy body?

CHAPTER V.

THE ADMISSIBLE VARIETIES OF PNEUMONIA.

Various modifications in the symptoms or seat of the disease—Pneumonia associated with acute rheumatism—Succeeding to pericarditis—Associated with renal affections.

Divergence of the disease from its assumed type.

WHILE pneumonia, as I have hitherto spoken of it, is a distinct and definite disease, it will not be supposed that any single description can include all its possible varieties. Like other affections it will be more or less. Certain of its symptoms may be unusually prominent, others masked, or suppressed, or variously modified. It is necessary only to acquire a clear conception of what constitutes the fundamental character of the disease in order to discriminate easily between its accidental and essential phenomena.

Modified forms of the disease.

In the foregoing account mention has already been made of certain modifications of symptoms which will readily be accepted. Such would be deferred or absent crisis, very rapid consolidation, the freedom from active pleuritic pain, a condition of prostration or of delirium, suggesting other diseases and withdrawing the attention both of physician and patient from the local malady. In a small proportion of cases the earliest symptoms do not even engage the lung, but rather the digestive organs ; sickness, slight jaundice, headache, vomiting, and sometimes diarrhœa, may precede the pain and cough and other characteristic signs.

Furthermore, certain indications on which we are taught to depend for an immediate diagnosis may be absent or altered. Thus, the proportion of blood mixing with the sputa and the mode of this admixture are subject to variation. Even in the purest pneumonia the sputum may by possibility be uncoloured, the smallest bronchules remaining unemptied by the feeble effort of coughing, or, on the contrary, the expectoration may be not only coloured but streaked and intermixed with pure blood, from the occurrence of actual hæmoptysis. Again, patients may be too simple or too weak to spit at all; the secretion is not wanting, it only fails to appear. In infancy, and sometimes in old age, we are prepared for this apparent departure from the rule of the disease. A concurrence of such anomalies, although no one of them at all invalidates the essential nature of the affection, may serve for a time to obscure it. It will be admitted, however, that pneumonia, like every other disease, must be capable of such deviations from any single example of it. It is less difficult to enumerate all its possible varieties than to make good the claim of any one of them to be regarded in the light of a type or pattern for the rest.

The same may be said of the local signs. We expect the earliest of these at the lower part of the lung: it is most often so. The upper portion of the lower or lowest lobe is, by a little, the most common starting-point of pneumonia. Yet it may have its seat elsewhere without forsaking its usual character; and we have no evidence that simple pneumonia attacking the upper instead of the lower lobe is distinguished by any special phenomena.

Pneumonia at the apex.

A marked lowering of vitality, a condition of the kind called 'typhoid,' attends indeed, as we shall see, all cases of pneumonia whatever which spread from point to point or from a number of centres at once. There are instances of the disease where, day by day, from the commencement to the time when the crisis becomes due, the area of lung involved obviously widens. These are marked by early and extreme prostration, and sometimes by the active delirium which everywhere is apt to attend such prostration.

Many quoted cases of apicial pneumonia are of this sort. The local affection starting in a central portion of lung does not make itself obvious to auscultation till it has reached its summit. The symptoms are modified, not because the apex is affected (for we know that the apex may suffer just like the base), but because inflammation commencing in the neighbourhood of the apex is easily overlooked. Instances of apicial pneumonia, therefore, are likely to be instances likewise of this spreading form of the disease, and it is this latter character, and not the accident of locality, which confers the peculiarity.[1]

Unusual prominence of nervous symptoms.

Again, we hear sometimes of 'cerebral pneumonia,' no more being meant than that nervous disturbance may be unusually prominent and unusually early, so as in some cases, and especially with children, to divert attention from the pulmonary condition. And, indeed, muscular spasm or violent delirium, or, with infants, repeated convulsions with a train of symptoms hardly to be distinguished from meningitis, may antecede the local signs of a quite uncomplicated pneumonia. I have elsewhere related the case of a young woman whose disease took the form of active maniacal delirium.[1] Dr. Farquharson quotes an instance[2] (the patient being a youth of eighteen) in which muscular rigidity passing into unconsciousness preceded the first notice of inflammation at the base of the right lung.

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[1] My friend Dr. Farquharson, of St. Mary's Hospital, in a paper (read before the Harveian Society, January 19, 1871) narrating and discussing several interesting cases of pneumonia, expresses the opinion that there is 'a special lowering of vitality attending extension of inflammation to the apex of either lung.' Admitting the first part of the sentence, I would venture to object to the limitation, and affirm that this lowered vitality attends *the pneumonia that spreads*, whether it reach the apex or not. I think Dr. Farquharson's own cases bear out that view. ('On Some Forms of Pneumonia,' &c.)

Such cases are most common in youth and childhood, owing to the instability, so to speak, of the nervous system in early life. As it is of the highest importance that they should be early recognised, an instance of this kind may be here interposed.

Case of delirium preceding pneumonia.

Thos. F., a light haired, strumous-looking child of six, was admitted into Westminster Hospital under my care on September 20, 1874, with a history of sudden illness commencing the day before on his return from school, when he complained of epigastric pain, and shortly became unconscious. On his admission the boy was in a state of extreme restlessness, throwing his head about the pillow, with widely dilated pupils, quite inattentive to attempts at arresting his attention, exhibiting, in a word, all the signs of active delirium within a child's reach. The temperature was 104·8, pulse 164, and respiration 41. It was with extreme difficulty he could be got to swallow. He continued in this state, with delirium and a temperature varying irregularly between 101 and 104, for three days, when on the 23rd (that is the fourth day of illness) small inspiratory crepitus with tubular breathing became audible at the base of the right lung. From that, though the temperature rose and fell during the next two days, the symptoms improved, and on the 29th (that is, six days later) distinct redux crepitation was heard, and the boy made a rapid recovery as from an ordinary pneumonic attack.

The knowledge that nervous symptoms of this kind sometimes develop into pneumonia should always be present to the mind, and, in childhood especially, may prevent a too early expression of opinion in regard to their origin.[1]

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[1] 'St. George's Hospital Reports,' vol. i., p. 343.

[2] *Loc. cit.* Case VI.

Secondary pneumonia.

But the recognition of pneumonia becomes really difficult when it occurs in association with other affections. It is impossible to maintain for it a separate place unless its relation to these is found to be consistent and orderly. If pneumonia is to be numbered in the category of diseases it must have a station as well as a name, it must be allied to some and incompatible with others. It will be necessary shortly to consider the nature of this relationship by observing the particular modes in which pneumonia occurs, or is alleged to occur, as a secondary or consecutive affection.

Its connection with rheumatism.

It has been mentioned already that among the many forms of pneumonia recognised by various authorities is the 'rheumatic.' Understanding the term as applying to pneumonia arising in the course of acute rheumatism, I would venture to urge that neither is the event sufficiently common nor the course of the pulmonary disease when so related sufficiently special to justify the admission of such a form.[2] Whatever we might be led to expect from a consideration of the pathology of the two affections and their point of junction, so to speak, in pericarditis, yet, in the guidance of mere clinical experience, it must be admitted that while rheumatic subjects of the proper age are more liable than their fellows to the disease in question, its occurrence in the midst of a rheumatic attack is not common, and that the appearance of this latter in the course of pneumonia is an event of extreme rarity.

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[1] It is of course competent for anyone to say the nervous disease *is actually*, at the first, the real and only one, be its associations what they may, the lung affection being secondary and consecutive, due to that special and, it may be, exceptional cause. What we are concerned with at present is not the relation but the order of appearance of the phenomena associated with pneumonia.

[2] Grisolle speaks of pneumonia as 'one of the rarest phenomena, at least in Paris, of acute articular rheumatism' (*loc. cit.* 425). He notices, however, that English writers do not describe it as very uncommon.

Is sometimes characterised by hepatisation.

There is, however, one peculiarity in the pneumonia of acute rheumatism which deserves to be pointed out, namely, *the frequency with which the first stage of the local process is pretermitted.* The lung is discovered to be solid without the preliminary crepitus having been observed. With considerable accord among authors as to the fact, there is much variety in accounting for it. It has been maintained that the absence of crepitus is due to the fact that the primary effusion is not in the alveoli but between them in the intervesicular areolar tissue, a statement which, even did it serve, is sufficiently disposed of, as Dr. Waters remarks, by the absence of any such tissue in that situation. I think that the true explanation is often suggested by the circumstances, as in the following cases.

Examples.

In the year 1864 a girl 19 years of age was admitted into St. George's Hospital,[1] and under my observation, for acute rheumatism of no great severity, and without heart-disturbance. On the second day she had a fit of alarming dyspnœa, with catching, shallow respiration, pain in the left side and in the affected joints. Repeated auscultation failed to discover any alteration of the heart's action, except in its increased rate. As little could any sounds be detected suggestive of pneumonia, though it must be mentioned that the patient's extreme dyspnœa rendered it impossible to examine the chest very completely. After remaining in great distress from breathlessness for two days, yet free from any mental disturbance, this girl died. On post-mortem examination the pulmonary artery, as far as its third and fourth divisions, was found to be filled with decolorised coagulum, and there was a shred of lymph in the right middle cerebral artery. The lower lobe of the left lung is described as 'much solidified from pneumonia, and sinking in water.' The heart was uniformly covered with recent lymph.

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[1] See 21 of Class III. Appendix D.

In the previous year a boy of 9 was admitted into the same hospital with acute rheumatism.[1] After eight days' residence he was attacked with 'double pneumonia.' The exact manner and symptoms of this attack I have forgotten. He recovered from it in a week. He next gets a return of pain in the limbs and alarming dyspnœa; the pulse rises to 120, and there is much visible pulsation of the carotids; free respiration, however, is still heard in the chest. This attack too passes off, and he is up and about again, well, or nearly well. When so far recovered, and with perfectly unembarrassed breathing, he has one night a sudden attack of angina and dyspnœa, which carries him off in a few hours. Here the whole of the right lung, except quite the apex, was red, solid, and airless, 'evidently,' it is written, 'in the first stage of pneumonia.'[2] The left lung too is affected in exactly the same manner, though less uniformly. The pericardium is adherent by recent lymph, the mitral valve very much thickened by fibroid matter.

Case in which pneumonia replaced the joint affection in acute rheumatism.

A third and more recent case is remarkable from the pneumonia appearing to replace the joint affection, and has sufficient interest to justify a fuller description.

William E., aged 28, carman, healthy, temperate, and well-nourished, but accustomed to winter cough, was admitted into the Westminster Hospital under my care on

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[1] Case 11 of Class VI. id. loc.

[2] See also a case by Andral, 'Clinique Médicale,' iii. 436, and one by Grisolle (426) of double solid pneumonia, associated with acute rheumatism.

January 30, 1874. He had been ill for two weeks from getting wet through and remaining so. Stiffness and aching of the limbs had gradually developed into acute rheumatism, affecting at last all the joints. It was the first attack of the kind, and we learnt that in the course of it he had 'fainted' more than once. On admission he presented completely the aspect of rheumatic fever; profuse acid sweating, flushed and tender wrists and ankles, universal immobility, a temperature of 102°, highly acid urine, and a tongue already dry and leathery. With the heart's sound no bruit or friction could be heard, but the normal area of superficial dulness was somewhat increased above. As for his progress, the temperature remained at about 103°, the pulse soft and weak at or about 112°; perspiration was profuse; he had some pain on coughing in his left side. No further physical signs were observed. Medicinally, he was treated with drachm doses of bicarbonate of potash in an effervescent draught, given at first and until the urine became alkaline (which it did on the third day), at frequent intervals, and afterwards every fourth hour. (On the recession of the joint symptoms a more stimulating treatment was adopted, which need not be detailed. The alkali was continued till near the end.)

On February 5 the joints had become, somewhat suddenly, almost free from pain, yet the man still sweated profusely, and the tongue was still dry. The urine was kept alkaline. The temperature was now 104°, the highest point yet reached. There was no marked dyspnœa or cough. The heart signs were not notably altered. The patient expressed himself as feeling much better, but except that his joints were set free, neither in his aspect nor general condition was any amendment indicated.

On February 8 (the tenth day) with a morning temperature of 103° (it had been declining daily since the fifth) and total absence of joint pain, though still bedewed with a sour-smelling sweat, this man became actively delirious. In that condition, during a very cold night of midwinter, he got up and partially dressed himself. Delirium of the same restless kind continued at intervals during the following day, so that he sought and sometimes gained opportunity of getting out of bed. On February 9, after a night of this sort, he was prostrate and exhausted, with a wandering mind which it was yet easy to recall. Some bronchial catarrh with slight mucous uncoloured spitting was attributable to the exposure; he had no return of joint pain. The temperature had fallen to 102·5°. After another night of restless delirium the patient rapidly sank, dying 12 days after admission and probably 16 days from the acute rheumatic seizure.

The post-mortem examination was made 30 hours after death, when the rigor mortis was well marked. In the pericardium there was about an ounce and a half of turbid fluid, with a few shreds of recent lymph. The heart weighed 13 ounces, its left ventricle was firmly contracted, the right ventricle was filled with blood clot. The whole of the valvular apparatus was competent, but a few small vegetations were found on both cusps of the mitral valve. The endocardium generally was opaque and somewhat mottled. The heart substance was soft but not degenerated, and there was slight atheroma at the origin of the aorta, the valves being healthy. The right lung was healthy, and weighed 18¼ ounces.

The lower lobe of the left lung was wholly hepatised, firm and unyielding, red passing into grey so as to give a marbled appearance to a section. It weighed 42 ounces. The surface was covered with thick shaggy lymph; the upper lobe compressed (presumably from fluid pressure), the pleural cavity containing much turbid serum. The other organs, all inspected carefully, were healthy.

Although not strictly in place, a word may be added here as to the pathology of such cases.

Pathological significance of such cases.

Without alluding in detail to the several steps in the inflammatory process, it may be stated generally that the condition which immediately precedes it, viz. hyperæmia and congestion, may be closely imitated by mechanical means. Venous obstruction, however produced, will give rise to an exudation which will be serous, or albuminous, or spontaneously coagulable, according as the pressure is less or greater. It is impossible, in short, to distinguish the hyperæmia due to this mere passive congestion from that which is due to commencing inflammation. It may be true that in the subsequent stages the inflammatory effusion, thus rudely imitated, has characters of its own which distinguish it from any that can be mechanically produced. It may 'tend to contain ingredients in larger proportion than that in which they exist in the blood; it may teem with organic life.'[1] It is unnecessary now to investigate these distinctions. Let it be conceded that inflammation is a process which cannot be exactly imitated by any mechanical contrivance. It is enough for our purpose that to the naked eye the resemblance is perfect;[2] that the changes which parenchymatous organs undergo, whether in size or increased solidity, or in appearance on fracture or on section, may be due to the one kind of infiltration as much as to the other.

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[1] Simon in Holmes's 'Surgery,' vol. i. p. 27.

[2] Dr. Robinson ('Med.-Chir. Trans.' vol. xxvi.) has recorded some experiments of his own upon the renal circulation, designed to show that the character of the exudation which takes place under pressure may be made to approach very nearly to that which occurs as a product of inflammation. By obstructing in various ways the flow of blood in the renal vein, this observer obtained not only liquid albumen and blood, but fibrin. He concluded that 'simple compression of the blood in its smaller vessels will, in a direct ratio to the degree of intensity of that compression, cause the exudation of an albuminous fluid, of coagulating lymph, or the extravasation of blood: its immediate effects, therefore, precisely resemble those of inflammation'—a result he would have been led to 'infer from the primary effects of inflammation being identical with those of and in compression of the blood, and the mere consequences of that physical cause.' The paper records the

If these observations be applied to the lungs, it will be at once allowed that the phenomena which attend mechanical obstruction elsewhere must occur with considerable modification in organs so constructed and charged with such functions. For it is by their means that the whole mass of blood is continually undergoing chemical change, in them this fluid, abundant beyond comparison of other organs, is at all points and without stop being brought into almost immediate contact with the external air, separated from it in fact by a membrane of such extreme tenuity that physiologists are still disputing about its structure. Consider that this vast network of capillary vessels is in connection, not, as elsewhere, with an areolar tissue to surround and support it, but with the air-vesicles ; or more truly, that the capillaries project into these cavities, their walls 'exposed and bare,' and with nothing save the thin membrane of the capillary itself between the air and the blood. In an apparatus so delicate, and charged with such functions, one would suppose that a very small disturbance, mechanical or otherwise, would suffice to produce changes, whether by exudation or hæmorrhage, such as would occur only rarely, and as an extreme result in the more compact and less blood-laden organs. If then we learn of the kidney, for instance,

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result of twenty experiments where artificial impediment obstructed the flow of blood through the renal vein. In the most striking of these, where the obstruction was incomplete, lymph was found in the bladder. 'I am not aware,' says the author, 'that any other instance is recorded of coagulating lymph as a consequence of simple compression of the blood by venous obstruction.' There follow fourteen further experiments, in which it was sought by various means to direct an increased flow of blood to one or both kidneys.

that by partial compression of the renal vein products are obtained which resemble nearly those which belong to inflammation, we should infer, I think, that such result would be obtained much more certainly and with much less inducement from any similar embarrassment of the pulmonary circulation. Still further, when it came to be considered that obstruction of a purely mechanical kind, rare elsewhere, is often obtained here by defect in the valves of the heart, we should be prepared to find that, in such cases more especially, exudation into the air-vesicles was by no means unfrequent. In a word, apart from the doctrine which would account for all morbid changes in organs by reference to inflammatory action, it would seem that consolidation of the lung might often be amply accounted for on mechanical grounds alone.

I would apply such considerations to the examples before us. It can hardly be doubted, I think, that all these cases are of one kind ; that they are instances of a sudden transudation of fibrin into the vesicular structure of the lung, the result, it may be, of but a slight exciting cause operating upon ready material—a weakened patient, namely, with blood in a highly fibrinous (or fibrogenous) condition, and a damaged organ wherewith to propel it. The final catastrophe depends upon the fact that circulation is only possible so long as the constituents of the blood, the obstacles presented to it, and the muscular power of the heart, hold certain relations to each other. When these limits are past, as in these cases, we might suppose beforehand that any accident which should diminish the force of the blood-stream on the one hand, or tend still further to increase the quantity of its fibrin-elements on the other, would be sufficient to give rise to a blood-stasis either general or partial, and so, by exudation, to a form of consolidation which should resemble, in the completest manner, both to the ear and the eye, the hepatisation of pneumonia.[1]

In the case of acute rheumatism, indeed, this transfusion of fibrin into the lung, in so far as it indicates the existence of hyperinosis, is analogous to pericarditis, and apt to concur with it. True, pericarditis is almost a natural consequence of undisturbed rheumatic fever, while lung consolidation happens to be rare in that relation; nevertheless both represent the same material result. The particular locality is determined by the genius of the disease.

Exceptional character of the symptoms.

An exudation thus rapidly poured into the pulmonary alveoli, and as rapidly consolidating, explains both the suddenness of the attack and the exceptional nature of the physical signs; it accounts also for the absence of characteristic sputa, a common observation in connection with the affection, and one which, in the ordinary routine of work as Registrar at a large hospital, I have repeatedly had to record. Owing to such declensions from the ordinary pattern of pneumonia, no less than to the fact that its proper pyrexia is merged and lost in the pyrexia of the acute rheumatism, attacks of this description (by no means necessarily fatal) are apt to be altogether overlooked.[1]

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[1] In the first volume of the 'St. George's Hospital Reports,' in a paper by Dr. John Ogle, there is a woodcut of a preparation which illustrates, as I conceive, the pathology of this form of consolidation. It exhibits a section of hepatised lung, with minute branches of the pulmonary artery filled with firm fibrinous coagulum. The specimen is from the Hospital Museum Series vii. No. 10, and is thus described in the Catalogue: 'Specimen showing red hepatisation of the lung, with extensive deposit of dark-red fibrin in the pulmonary artery. Double pleuro-pneumonia had existed, and there was tolerably firm adhesion between the layers of the pericardium; the cavities of the heart were dilated, especially the left auricle, which was lined by recent yellow fibrin. The margins of the mitral valve-flaps were occupied by recent fibrin also, and slight atheroma of the root of the aorta existed.' See vol. i. 'St. George's Hospital Reports,' p. 168.

Unmodified pneumonia in acute rheumatism.

It is not to be supposed from what has been said that pneumonia in acute rheumatism always happens in this fashion. On the contrary the physical signs which first announce it are often the same as when it occurs alone. Yet even so the special characters of the consecutive disease, in its mode of access and sudden critical remission, are necessarily disfigured by the coexisting rheumatic pyrexia. It is not possible, therefore, that pneumonia in this conjunction should appear altogether in its usual dress, so as always to ensure recognition. Yet, subject to these modifications, it follows the same course, and its resolution is accomplished by the same stages as have already been described for the simple disease.

And not only in the clinical features does this pneumonia declare itself as such, it may be recognised as well by corresponding anatomical characters. The solid, red or marbled, lung of liver-like consistence, and with a surface covered over with recent lymph, as in the cases just detailed, is emphatically, as we shall presently see, the lung of pneumonia, and recognisable as such by the naked eye.

Occurrence of pericarditis.

In this coincidence of rheumatic fever and true pneumonia the occurrence of pericarditis may be referred to either disease. Yet sometimes, without rheumatism, or at least without its common symptoms, an attack of pericarditis may be followed by one of pneumonia, as though the two inflammations proceeded from some common origin.

Thus a man of twenty-seven, not intemperate, was admitted into the Burdett Ward of the Westminster Hospital, on April 30, 1875, with pericardial friction and moderate fever, but with neither joint inflammation nor acid sweats. He had long complained of pain in the head and chest, had been much worse during the last two months, and kept from work a week. The rubbing shortly subsided, but not the increased area of cardiac dulness, and on May 18, though imperfectly recovered, the man went out. He returned, however, two days later, having had a rigor in the interval with sharp pain in the left side. His pulse was now 116, and respiration 36, and temperature 104·6, the highest point reached.[1] The physical signs now indicated consolidation of the left lung. Total absence of vocal fremitus, with some quavering of the voice, indicated at the same time the presence of fluid. The general symptoms were those of pneumonia, yet the aspect was not characteristic, and there was more restlessness and active movement than is usual. On the day before death, which took place on the sixth day of the lung affection, loud pericardial friction became audible, due, as clearly appeared, to renewed contact of roughened surfaces. The man became livid and slightly jaundiced; the urine albuminous. He died asphyxiated.

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[1] The more so inasmuch as in such association pericarditis is likely to be more obvious as well as more present to the mind than either pleurisy or pneumonia. Should chest pain be complained of at all amid such grievous pain everywhere, it serves to direct attention to the heart rather than the lungs, and here the discovery of friction seems to account easily for the symptoms without seeking further.

Case of pericarditis succeeded by pneumonia.

The post-mortem examination, besides exhibiting grey hepatisation of the lower lobe of the left lung, showed on that side a thick layer of pleural lymph. This membrane was closely adherent to the ribs; externally it was tough and almost leathery, but next the lung puro-fibrinous and soft like butter. The extreme base of the lung had escaped consolidation, and in the pleural cavity was about half-a-pint of straw-coloured serum. The pericardium contained a like quantity of fluid; its surface was rough like a nutmeg-grater, and displayed the peculiar reticular arrangement often seen in such cases; some recent shaggy lymph adhered to it, but the surfaces were nowhere in union. The right lung was healthy, and no other organ notably diseased.

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[1] It is observable that although this patient died, and from the first gave no sign whatever of amendment, his respirations rising to 52, the temperature underwent, what would be called from its chart, a critical fall. Reaching its highest point, 104·6, on the second day, it fell continuously till the fifth, when it was below 100. The day before his death, when the pulse was 148 and respiration 52, the temperature was 100·5. Dr. Peacock records the same thing, see Appendix B.

Anatomical inspection thus bore out the clinical history, and showed pneumonia as the last in a chain of morbid events which, commencing probably with the pleura two months before, first took the form of active disease with the occurrence of pericarditis, the pneumonia arising upon this, in what precise connection it is not easy to determine. A limited amount of pericarditis is, I believe, far from uncommon in pneumonia, and often passes unnoticed. As post-mortem evidence amply supports the opinion that a larger number of persons suffer pericarditis than have it recorded in their lives, so also does the clinical character of that disease, and the fact that a rubbing sound, itself an accident, is sometimes its sole objective sign. In sixteen fatal cases of simple pneumonia there is pericarditis in six, or more than a third.[1]

Pericarditis not uncommon.

There are, it is probable, other varieties of associated pneumonia, besides the rheumatic, determined by specific blood changes.[2] But the claim for the disease to be so regarded in any particular case must not be admitted in these general terms or without explicit restriction. For it is just at this place, and by the use of vague expressions, that pneumonia gets mixed up with hypostatic congestion, and thus loses its place as an individual disease. It must be understood, therefore, that we are not speaking now of that insidious form of pneumonia which, as is alleged, 'grafts itself' upon various disorders of the most opposite kind. For myself I fail to recognise such an affection, nor can I conceive of any definition that could by possibility be framed for a single disease, and yet include so much.

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[1] *Vide* Class IV., Appendix D.

[2] There are, no doubt, certain conditions of the body—conditions in themselves departures from health—which render the individual more exposed than usual to the attacks of pneumonia. We have seen that the disease is remarkable among other things for the suddenness of its onset. Nevertheless, that bodily state which renders an apparently healthy person, at a particular moment, assailable by pneumonia upon some slight provocation—which puts him, for instance, at the mercy of an east wind which he has faced with impunity a hundred times before—is in itself a condition of disease, or of unstable health equivalent to disease. Now the condition supposed may be either an accident of

Imperfect urea discharge in kidney affections as predisposing cause.

There is not wanting direct clinical evidence to prove that imperfect elimination of urea is to be reckoned among the causes which predispose to true lobar pneumonia. The other factors being present, defective secretion on the part of the kidney, (whether from chronic degeneration and wasting, or the occlusion of uriniferous tubes, or, perhaps, some sudden and temporary suspension of the kidney function due to exposure,) may have this amongst other consequences. Thus, it will be seen from the Appendix that renal disease figures largely among the pre-existing morbid conditions of the individuals enumerated. And it is to be observed that in those instances where it seems most feasible

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the moment or it may have lasted long enough to have attracted notice. In the one case the resulting affection is spoken of as idiopathic, in the other as secondary or consecutive. The distinction is necessarily inexact and artificial, it depends upon whether this antecedent condition has escaped or has obtained separate recognition. The disease is named according to the duration and manifestness of that abnormal condition which in all cases alike must precede its attack. Hence the origin of idiopathic pneumonia may be similar to that which we call secondary. Similar yet not identical; duration is an element of difference. A sudden derangement in elimination may be more readily or more acutely resented than one which is more gradual. Yet in both instances the same cause is in operation; in both it is the excess or the defect of the very same discharge that gives occasion for the new morbid action.

to suppose that the one condition is a consequence of the other, the pneumonia is not deprived by this association of those features which commonly distinguish it.[1]

Liability to pneumonia of the several forms of renal disease respectively.

It appears upon calculation that pneumonia in kidney disease is most frequent in the so-called amyloid or waxy degeneration. According to Dr. Dickinson the marked tendency to inflammation which characterises this change is seen most conspicuously in the lungs and next often in the pleura. Tubal nephritis, on the contrary, is associated with pericarditis and pleurisy more often than with pneumonia, while in granular degeneration (where the commonest inflammatory affection is bronchitis) neither pleurisy nor pneumonia is often met with.[2]

Difficulty in detecting co-existing renal disease.

It is not always easy to interpret aright the indications afforded by the urine. Albuminuria due to renal congestion we have seen to be of common occurrence in pneumonia, almost a part of its natural history. It is necessary, therefore, before accusing the kidneys, to ascertain the precise significance of this particular symptom, a task which may be difficult or impossible so long as active congestion continues.

Indications furnished by the urine.

A large amount of albumen or the presence of certain casts of the uriniferous tubes are signs of pre-existing renal disease. For although the urine may rarely contain traces of blood, and moulded casts of blood corpuscles, and even of fibrin with broken epithelium (the condition of the kidney resembling in fact that of the lung itself), yet continued hæmorrhage or the repeated occurrence of epithelial or finely granular casts is an evidence of kidney disturbance which it is beyond the power of simple pneumonia to provoke.

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[1] As, for instance, in Cases 2 and 3, Class II., Append. D.

[2] See Dr. Dickinson, 'Pathology &c., of Albuminuria,' pp. 191–2. Of course these readings of statistics fail to show precisely the liability to pneumonia which these affections respectively confer. They require correction in reference to the fact that pneumonia is a disease of early adult life, and thus likely to concur (even when arising from independent causes) with the forms of kidney disease proper to that age, with tubal nephritis, that is, rather than with granular degeneration.

Pneumonia simulated in renal disease.

There is another source of error. Renal disease may be ascertained beyond question, and, along with general anasarca, auscultation may discover small crepitating râle. Let it be remembered that in such a case pneumonia is not proved without further evidence beyond that which is afforded by this sign. Œdema of the lungs, like œdema of the limbs, is very apt to overtake such subjects, and from this likeness of sound, to be mistaken for active inflammation. The crepitation of pulmonary œdema may approach very nearly to that of commencing pneumonia. The occurrence of crepitating rhonchus is, of itself, as has been mentioned, of doubtful significance. In an œdematous patient, heard at the base of one or, much more, of both lungs, it must be taken to indicate not a new disease, but the extension to these organs of the existing dropsy.

Hypostatic consolidation mistaken for pneumonia.

It is necessary, moreover, to separate from pneumonia the consolidation (to be described presently) which is apt to follow upon such œdema, and is associated, therefore, with chronic heart and kidney affections. Insidious in origin and of indefinite duration, this hypostatic solidification is far more common than the acute disease for which it is often mistaken. This latter for the most part attacks the subjects of kidney disease as it attacks others, only probably with somewhat more frequency.[1] It does not steal

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[1] It may be asked what amount of kidney change observed after death is sufficient to prove that the pneumonia is consecutive? How is the distinction to be made between pneumonia arising directly from imperfection of the kidney, and the same disease arising independently in an individual possessing that imperfection. It is obviously impossible to make it, and for this reason, no less than from the difficulty already mentioned of appreciating at the time the exact bearing of albuminuria, statistics upon the subject are encompassed with difficulty and error. If we choose to refer pneumonia to imperfect elimination, most kidneys will serve for illustration, since in and after adult life these organs are

Purulent conversion of the lung favoured by kidney degeneration.

upon them unobserved in the midst of their other troubles, or attach itself secretly to an already engorged and inactive lung. Pneumonia under such circumstances is impossible.

Yet the presence of kidney disease imports a special danger. Patients with this defect are thereby rendered liable to that form of pneumonia which ends in destruction of the lung by suppuration. For them, therefore, the disease, although it may seem to follow its usual course, is always to be regarded as having a gravity of its own, while conversely, every instance of pneumonia tending to this end should suggest inquiry as to the state of the kidney.[1]

This fatal event of pneumonia, however, is seen as well in other relations, and will be best discussed when these have been considered.

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seldom absolutely healthy; while on the other hand, the argument that pneumonia occurs usually as an intercurrent, independent disease, the kidney defect notwithstanding, receives support from the open and orderly character of its symptoms when seen in this particular association.

[1] See a paper with cases in the 'Dublin Medical Quarterly,' May 1856, by Dr. McDowell. It will be seen that the instances therein adduced—some of them very characteristic—are not *all* instances of pneumonia according to my definition of it.

CHAPTER VI.

ADMISSIBLE VARIETIES OF PNEUMONIA—*continued.*

Alcoholism determining the course and event of pneumonia—Delirium tremens masking the proper symptoms of the disease—Purulent invasion of the lung associated with privation—Asserted connections of pneumonia—with specific fevers—with defective hygiene.

Intemperance and famine influencing pneumonia.

NOT in renal disease alone does the lung exhibit, in a marked manner, the tendency to rapid destruction by suppuration. There is a class of cases—difficult both to recognise and to classify—where apart from any lesion on the part of the kidney, life is destroyed in the same manner. I allude to instances where pneumonia attacks those who are addicted to alcoholic excess or weakened by want of food and the other privations that belong to extreme poverty.

Insidious course of the disease.

Not rarely, for example, a patient under treatment for delirium tremens will rapidly succumb, as it seems, from exhaustion produced solely by that disease. On post-mortem examination, however, one lung will be found extensively consolidated and infiltrated with pus. Less often an individual, not a drunkard or a tippler, with undefined symptoms ascribed to privation and exposure, or with those signs of prostration which, during an epidemic of the kind, are so readily attributed to continued fever,—will suddenly die, not from fever, as it turns out, but from purulent infiltration limited to one lung. Instances of the kind take us by surprise. Often it is only discovered *post mortem* that any lung affection exists. Yet it argues no great want of vigilance that such extensive mischief should have been overlooked in the patient's lifetime. It is so rapid and stealthy, that in some cases a careful exploration of the chest shortly before death has failed to discover any sign of lung consolidation. In others the fact that the patient was about his work nearly to the time of his death excludes the supposition that acute pneumonia has run its course to a fatal termination by the usual stages.

Special character of such pneumonia.

It is beyond question that cases of this sort have from the very first a character of their own. With extreme prostration the local affection may be limited in extent, and, as sometimes with typhus, the pulse infrequent.[1] These are not instances of a benign disease proving fatal owing to want of treatment, or the already enfeebled condition of the person attacked. They exhibit a morbid energy of their own which seems beyond the influence of drugs, and of which death is the natural and probably inevitable end. Yet while they belong to a distinct class they are not to be excluded from the category of pneumonia unless we determine to limit it by further restrictions than have hitherto served.

Delirium tremens masking its symptoms,

As for the alcoholic class it is matter of observation that this particular poison favours the purulent conversion of an inflammatory exudation.[2] Given a patient of the kind supposed, it is easy to see that the symptoms of alcoholism, especially that form of delirium which is at once so readily accepted as a disease of itself, would mask the symptoms of co-existing pneumonia. With no extreme pyrexia or dyspnœa, even though the whole of a lung should be involved, the patient —often without cough—is insensible to the pain of stitch and incapable of the act of spitting; he offers no symptom which suggests or seems to require any further investigation. It is conceivable, I say, in these circumstances, that true active pneumonia should run its course unperceived and unsuspected. If, in such a case (and there are many such), upon the occurrence of death the morbid appearances are similar to those usually found in purulent infiltration, the pleura being implicated along with the lung, I know not upon what theory the disease is to be separated from pneumonia, or in what respect its clinical history is opposed to that view. The fact that its presence is sometimes thus obscured only teaches the importance of a frequent examination of the chest organs in all cases of the kind.

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[1] Trousseau records in such a case a pulse of 84 with a respiration of 88; 'English Trans.,' vol. iii. p. 358.

[2] See a paper by Dr. Dickinson on the morbid effect of alcohol, as shown in persons who trade in liquor, in 'Med.-Chir. Trans.,' vol. lvi. p. 27.

or modifying its course.

And just as this association of a special form of delirium will suffice sometimes to conceal the initial symptoms proper to pneumonia, so at any period of its course the same phenomena may intrude. A case may be characteristic at the beginning and obscured towards the end, so that it shall be given this name or that, according to the accident of its being seen earlier or later.

Case of alcoholic pneumonia.

Some years ago my friend Dr. Bagshawe, of St. Leonards, gave me the opportunity of seeing a marked instance of this. The patient, a woman of forty, was of intemperate habits, and had been living upon meagre diet. At the time I first saw her she had a moist, cold skin and aspect of great prostration. During the previous night she had been wildly delirious, tossing about the bed-clothes, and difficult to restrain. She had also had tetanic spasm, affecting chiefly the facial muscles. There was neither cough nor expectoration. Now this woman's attack, as I learned from Dr. Bagshawe, had commenced, seven days before, with rigor following exposure to wet. She had had stitch and cough, and rusty sputa, and when first seen on the fifth day of the illness tubular breathing was discovered, with dulness over the lower three-fourths of the right lung. The day after my visit she became apyrexial; but the mental disturbance continued, and she was at times difficult to restrain. In the meanwhile the lung underwent the usual changes of resolution. Long before the final cessation of delirium, and when the tongue was still dry, and the patient disposed to be violent, redux crepitation appeared, shortly followed by natural breathing.

Starvation pneumonia.

But cases occur also which are not accounted for on the ground of alcohol poison—cases of mere want and privation, terminating, as it would seem on that account, in this purulent conversion. Such pneumonia gets associated with typhus, not from any inherent likeness, but because it puts on some of the features of that affection, so as to be mistaken for it. The best illustrations of this form of the disease occur in middle or advanced life, and are met with away from hospitals, often amongst those whose deaths have been so sudden as to render an inquest necessary.[1]

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[1] By attending such inquests my friend and colleague Dr. Robert J. Lee some years ago obtained the particulars of several cases of sudden death which he was kind enough to place at my disposal. From the number I quote the following:—

CASE 1.—*Purulent Infiltration of the lower Lobe of the left Lung.*—March 11, 1872. An inquest was held before Mr. Bedford on the body of a woman, 55 years of age, of dissipated habits, who had been found dead early in the morning in her bedroom. She had come home the previous evening as well as usual, and had retired to bed without complaint. She was kneeling at the bedside when her room was entered in the morning, and had been dead but a very short time. The only disease that was discovered on examination was universal grey hepatisation of the lower lobe of the left lung; all the other organs were healthy.

CASE 2.—*Purulent Infiltration of the upper Lobe of the left Lung.*—A respectable man, aged 48, died unexpectedly in his bed, and an inquest was necessary. For three years he had worn a tube in the trachea in consequence of laryngeal disease, for which he had undergone tracheotomy. He had been unwell for two days previous to his death, but not so seriously as to excite any apprehensions. The upper lobe of

Lung consolidation having this origin not always pneumonia.

All such cases, as I believe, are divisible into two classes: the one pneumonia, the other not. In the first, the stages of the real disease are very rapidly fulfilled: lowered vitality of the patient operates to accelerate its ordinary changes and render them immediately destructive. Clinical phenomena are not wanting—they are only abridged; while the poverty and neglect, which are here essential factors, do not consist either with immediate attention to this ailment on the part of the patient, or early opportunity for observation on that of the physician. From first to last, even with a very extensive implication of lung, there need be no extreme temperature. Yet the condition revealed after death is essentially pneumonia, differing in nothing from that of a patient who, by the most orderly steps, has survived long enough to reach the same stage. Anatomically, that is to say, the disease is unilateral and sometimes apicial; it is strictly limited; there is little or no accom-

the left lung was entirely solid from grey hepatisation. The disease was limited to this part, and there was no evidence of inflammatory changes in other parts of the lungs. All the other organs of the body were healthy.

CASE 3.—*Purulent Infiltration of the whole of the right Lung.*—An examination was made on May 1, 1872, of the body of a man who had disappeared, without any reason, about ten days before, and who, it was proved by the evidence adduced at the inquest, had committed suicide by drowning in the Thames. He was a middle-aged man, and had been attending at Guy's Hospital as an out-patient for a few days before his death. On the day his death occurred he had been driven to the hospital, and had left his sister's house late in the afternoon with the intention of walking home. Nothing was heard of him again till his body was found in the river. The whole of the right lung was in a state of grey pneumonia. The left lung and the bronchial tubes were quite free from any of the usual appearances of acute inflammation. The other organs were healthy. It was suggested that in a state of mental derangement, a not unfrequent symptom of pneumonia, the act of suicide was committed.

It is unfortunate that the condition of the pleura is not mentioned in any of these cases.

panying bronchitis; the pleura is invariably implicated. The heart and other organs are often healthy.

Destructive alveolar catarrh to be distinguished from it.

In the second class of cases (though the distinction may not always be suffered to appear, owing to an imperfect history, or partial and inexact post-mortem inspection), the disease is but the final stage of a prolonged illness, of which the general character and consequences may be gathered from inspection of other parts of the body: dwindled, granular kidneys with diminished cortex, a thin and dilated right ventricle, often encroached upon or changed by fat: not in one but throughout both lungs evidence of bronchitis; while the organ chiefly affected, though solid, is yet compressible, and, carefully looked at, is seen to be consolidated by lobules or collections of lobules, exhibiting, between these irregular tracts of mere congestion, a condition which it shares with the other lung. Both are, indeed, similarly affected, though in different degrees, insomuch that such cases are often described, living and dead, as 'double pneumonia.' There is seldom pleurisy,[1] but often one or both pleural cavities contain fluid. The subject of this condition

'Double pneumonia' often of this form.

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[1] Does true pneumonia ever occur 'as the sole disease' without the accompaniment of pleurisy? Dr. Stokes believes that it does. The evidence of twenty years of hospital reports (the hospital being St. George's) supplies no undoubted case of such an occurrence, and but one (Case 15, Class v., Append. D) which can by possibility be so interpreted. It is as follows:—A woman, aged 36, was admitted into St. George's Hospital, after a week's illness, of which the first symptom was shivering and stitch in the right side. About the same time she got cough and dyspnœa. When first seen, she had a frequent pulse, hot and dry skin; the pulse was 120, respirations 52, the tongue dry and brown. A few moist sounds were heard at the apex of the left lung; the breathing generally was harsh and imperfect; there was neither ægophony nor tubular breathing. The case was at first regarded as one of pneumonia, and treatment by antimony was adopted. On the second day, however, though the respiration was less hurried, and there was little cough and no expectoration, delirium had set in, and an ill-marked mulberry-rash was visible. The chest-sounds were less marked than at first. The patient was now supposed

has suffered many previous attacks similar to that which at last kills or helps to kill him. Variously named, it is now commonly called broncho-pneumonia. Doubting its right to that title, I would exclude it from the admissible varieties of the affection we are discussing, and mention it now but for the sake of comparison. I shall endeavour to show in its place that the points of resemblance to pneumonia are apparent and not real, and that the older physicians were guided by a true instinct in speaking of it as false pneumonia.

Pneumonic consolidation as a pathological condition separable from pneumonia as an active disease.

There is another form of hepatisation. It is found in instances where aneurismal or other tumour, or fibroid thickening, exerts long-continued pressure upon the lung, or implicates especially some portion of the pneumogastric. Long ago the well-known experiments of Reid upon this nerve in the lower animals showed that its section was followed first by hyperæmia, and soon by consolidation of

to be suffering from fever, and in that view the treatment recommended by Dr. Dundas was followed—that by large doses of quinine at frequent intervals. In this way more than eighty grains of quinine were swallowed in twenty-four hours, by which time the skin was quite cool and the pulse had fallen to 72. At the very instant that these particulars were being noted down, the colour faded from the woman's face and she immediately expired, having been under observation for about four days. In this case it was found that nearly the whole of the right lung was in a state of grey hepatisation. The pleura was natural, both ventricles of the heart were uncontracted, and the blood was fluid.

Without arguing that this patient must have had fever because she was treated for it, it must be allowed the history of the case corresponded in many points with that of fever (typhus was prevalent in the hospital at the time), even to the appearance of a mottled eruption; while there was nothing in the post-mortem examination to negative that view. On the other hand, if the case be regarded as one of simple pneumonia, latent during the greater part of its course, it is, so far as these records go, a unique case; for in no other does simple pneumonia occur without the accompaniment of pleurisy. In other words, in every case where lung-consolidation, both from its pathology and history, may not reasonably be looked upon as secondary, there is pleurisy going along with it.

the corresponding lung. That such a result does not uniformly follow when one side only is dealt with, may be due (as Sir William Gull suggests) to the intricate union through the pulmonary plexus of the nerves of the two sides, by means of which, defect of the one is supplemented by the action of the other. Section of both pneumogastrics, it is affirmed, invariably gives rise to an exudation into the pulmonary alveoli, while, if the animal live long enough, the consolidation thus accomplished will appear in detached patches which eventually break down into purulent cavities. 'In forty-eight hours,' says Dr. Pavy, 'I have noticed the lung-tissue broken down into small purulent cavities.' In the case of disease—of aneurism especially—a result somewhat similar to that obtained by sudden section is attributed to the gradually increasing pressure of the tumour, a pressure not exerted on the upper portion of the pneumogastric, but, lower down, upon one of its branches.

A chronic condition, which may remain stationary for long periods,

Now, to whatever extent this theory may be applicable, the cases to which it refers—of aneurism or cancer, or fibroid thickening—though they may exhibit hepatisation at death, do not in life exhibit the symptoms of pneumonia. The patients are not suddenly seized with an acute affection in the middle of their other suffering. On the contrary, lingering on indefinitely with symptoms attributable to the original disease, and muco-purulent blood-stained and sometimes fetid sputa, they die at length with some portion of lung solid, or diffluent, or gangrenous. Or, if it should happen that during life the physical signs on the part of the lung have occupied attention, these are ascribed, as a matter of fact and history, to chronic changes, to phthisis, to chronic pleurisy, to pulmonary collapse.

is apt to slough and become gangrenous,

Pathologically, this condition is marked by the absence of pleurisy; by the patchy character and unequal progress of the several parts of the consolidation; and by its tendency to remain stationary for long periods, or else to slough and run into gangrene. Such changes, aided by the mechanical effects of pressure, are probably the direct result of a paralysis of those parts of the bronchial track which are deprived of their nervous supply. In turn, accumulation of secretion, dilatation of tubes, exudation (the result of obstruction), and finally, disintegration from arrested nutrition, are the several steps in a process which has its strict analogy in other parts.[1]

resembling in some respects lobular collapse.

These phenomena are separated widely enough from those of ordinary pneumonia; they resemble, in certain respects only, that destructive purulent form of it of which I have just spoken. In rate of progress and marked liability to gangrene, this state of lung approaches nearest to what we shall have to observe presently in connection with lobular collapse.

While, therefore, there is reason enough for separating this secondary condition from pneumonia, it cannot be excluded altogether from the category of inflammation. I would speak of it as *pneumonic consolidation*: pneumonic because the process of destruction through all its stages is obviously strictly conformable to what occurs in destructive inflammation elsewhere; not pneumonia, because the manner of that particular inflammation is *sui generis*, progressive from beginning to end without halting, not usually destructive, and never terminating in gangrene.

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[1] See a paper by Sir William Gull on 'Destructive Changes in the Lung from diseases in the Mediastinum,' in 'Guy's Hospital Reports,' vol. v. Of the three cases quoted one was aneurism, one cancer of the œsophagus invading the trunk of the right vagus and branches of the pulmonary plexus, and one fibrous thickening around the right bronchus with similar implication of the right pneumogastric; there was gangrene in the second case, a huge sloughing cavity in the third, and in the first the implicated lung was irregularly consolidated.

In Appendix D, Case 25, will be found an instance of solid lung from the pressure of a distended pericardium. The illness here commenced with pneumonia of the *left* side, which recovered, and in the subsequent pericardial effusion this secondary implication of the *right* lung passes unnoticed. The case thus affords a striking illustration of the difference between consolidation and pneumonia.

Asserted connection of pneumonia with continued fever

Many other varieties of pneumonia are current which, for reasons that will presently appear, I would also exclude from this enumeration. It is common, for instance, to hear of the disease complicating continued fever, and especially typhoid. Every work upon fever contains directions for detecting and treating it in that relation. And, undoubtedly, both in typhus and typhoid the lungs sometimes become solid. Such an event may even contribute directly to the death of the patient. Nevertheless, the appearance of this solidity, when it comes to be inspected, rarely bears out the theory of intercurrent pneumonia. The sequence is less spoken of and less known in the post-mortem room than in the wards. My own belief would exclude pneumonia altogether from the history of these affections.[1]

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[1] In regard to the relation between pneumonia and the continued fevers, the statements of authors would seem to warrant the conclusion that the concurrence of the two diseases is not rare. Dr. Murchison noted it (that is, clinically) in 13 out of 100 cases, and Flint in 12 of 73. The lung affection is believed to be commoner with typhoid than typhus. But what is the anatomical character of this pneumonia? In typhoid Dr. Murchison says 'it is usually lobular.' 'In rare cases the circumscribed condensed patches appear to become converted into small abscesses, or they pass into gangrene.' Two illustrations are given of the discovery of pneumonia *post mortem* in cases of typhoid fever. In the first, a boy of eleven, it is 'lobular, isolated nodules are scattered through *both lungs*, the tissue of which was very hyperæmic, friable, and scarcely crepitant, but not granular on section.' The right lung weighed 12, the left 13 ounces. *The pleura is not mentioned.* In the second case, a youth of 19, *both lungs*, but especially the right, contain a number of circumscribed nodules of granular consolidation, 'varying in size from a pea to a walnut,' the right lung weighing 30, the left 24 ounces. *Again the pleura is not mentioned.*

Such being the mode of pneumonia in the form of fever which is most obnoxious to it, we read further that in typhus 'true pneumonia is rare, and commonly lobular. It is not always possible to distinguish pneumonia during life from pulmonary hypostasis, and *in fact the lesions may exist together.*' In the rare instances in which pleurisy is observed and scarlatina.

As for the connection of pneumonia with scarlatina, it is certainly not intimate.[1] On the other hand, the relationship between it and measles will be near or remote, according to the view taken of the nature of lobular consolidation as it occurs in young children. The point may be reserved for future consideration.

'Pythogenic pneumonia.'

I must not so dismiss a form of so-called epidemic pneumonia intimately allied to enteric fever and occurring under like circumstances, from sewer gas, imperfect drainage, and like causes. Undoubtedly, symptoms of chest inflammation with both physical and general signs resembling pneumonia do arise from the causes alleged, and sometimes prevail epidemically. It has been proposed to call the condition 'pythogenic pneumonia.'[2]

In these cases the patients exhibit all the phenomena of fever-poison in prostration and pulse, together with those of active pulmonary congestion, attaining sometimes to consolidation. There is an absence of pleuritic stitch, often there is no expectoration, some bowel disturbance is common, there is apt to be diarrhœa with sometimes ochre-coloured stools. The lung affection is often double, and in many instances its physical signs, even at an advanced period of the disease, amount to no more than fine crepitation. When these cases end fatally, death is not from the local but the general affection, and the manner of the patient's sinking is often strikingly suggestive of typhoid. On post-mortem inspection, too, the lungs are not hepatised

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in typhus 'its advent is latent. The effusion is almost always fluid.' 'When pneumonia does occur it occasionally terminates in gangrene.' There is nothing in these statements which at all conflicts with anything I have stated. The difference resides in the nomenclature. See Murchison, 'On Continued Fevers.'

[1] Dr. Gee states that lobar pneumonia sometimes occurs in scarlatina. See his art. 'Scarlatina,' in 'Reynolds's Medicine,' vol. ii. 344.

[2] Drs. Grimshaw and Moore, 'Pythogenic Pneumonia,' in 'Dublin Journal of Medical Science,' May 1874.

but unequally solid, or solid in patches. There is no recent lymph upon the pleura, and no accompanying pericarditis.

Its points of difference from true pneumonia.

The subjects of this form of disease are often those who have been exposed to the very same influences which have sufficed at the same period to impart enteric fever to others their fellows. They are, in fact, the victims of the same poison, and the pulmonary congestion which is but one indication of their suffering is seldom the chief source of it. If this condition, which so nearly resembles that which arises in connection with typhoid, is to receive a special name indicative of its origin, it should be called rather pythogenic lung congestion. The consolidation is sometimes hypostatic, and sometimes it is associated with collapse and found in detached nodules.

Finally, let it be granted that there are many diseased conditions affecting secretion in various ways, each of which must have a place of its own as respects the liability to pneumonia which it confers. Our insight is here very limited and uncertain, yet besides what actual observation warrants, the ingenuity of authors will ever be contriving new forms and new relations of the disease. In determining the general canon which is to judge them, we must be mindful that the same sharp line of difference which by our definition would seem to separate pneumonia from its counterfeits, will be lost now and then in actual practice. There is an ill-defined middle territory which, if it cannot belong rightly to both, must yet remain the subject of a disputed ownership. While, therefore, here as elsewhere, room must be given for anomalous and exceptional cases—since a perfectly rigid classification in practical medicine is secure of failure—it is yet possible, as I believe, to lay down with some precision that which constitutes necessarily, so far as experience serves, the objective phenomena of pneumonia.

CHAPTER VII.

MORBID ANATOMY OF PNEUMONIA.

Morbid specimens illustrate the disease incompletely—The anatomical stages of pneumonia—Histology—The process of 'resolution.'

The early stages of pneumonia rarely met with anatomically.

In attempting to investigate the nature of any disease by reference to morbid anatomy it is necessary to bear in mind, as determining the value of such evidence, the precise circumstances in which this particular method of inquiry becomes available. Pneumonia, as we have seen, belongs to a class of affections which are not as a rule fatal to life Death occurs now and then, but it is exceptional and due to accidental causes. In the most characteristic instances of the disease, therefore, the opportunity of making actual inspection of the organs chiefly implicated does not arise. Even in the small number of cases where death results directly and immediately from pneumonia, the event will be likely to occur late and to reveal no more than the final result of a long series of morbid phenomena. The changes that accompany commencing hepatisation or the process of resolution, since they concur with a vitality which only some rare accident can suddenly arrest, are not of a kind to be often seen. As a rule it is only when the acute affection has spent itself, and its early anatomical features are obliterated, that we are permitted to inspect its ultimate results and the place it once occupied. The patients that die, die too late to afford fair illustration of the active disease,

Post-mortem specimens likely to be defective.

while those that recover—the typical and characteristic cases—so far as pathological specimens are concerned, are not represented at all.

The disease anatomically to be recognised by the aggregate of appearances.

It must be observed, however, that physical conditions similar to those through which the lung passes in the course of pneumonia, are to be met with elsewhere in circumstances more favourable for post-mortem observation. The anatomical features of the affection are not expressed by any single appearance, but by an assemblage of phenomena which tend to explain one another and acquire a definite significance by their concurrence. Just as in the living patient there is no one symptom which of itself certainly indicates pneumonia, so in the dead subject it is not by separate signs but by regarding these in the aggregate that the nature of the disease is determined. It may even happen that the post-mortem evidence is of itself insufficient until read by the light of the clinical history, or, conversely, that ambiguous symptoms during life can only be interpreted by the revelations of death. It is thus impossible to dissever the clinical from the anatomical part of the disease, since one and the same appearance may be credited with several meanings according to its living history.[1]

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[1] It is the custom in systematic treatises to describe the lung condition from the earliest hyperæmia to the final consolidation, as though through all its stages the information derived from auscultation were of equal validity. It is obvious on reflection that such is not the case. For example, there are many kinds and degrees of consolidation, and the ear can never distinguish between them. So long as the lung is airless as to its vesicular part, and the material that occupies it is not stirred by the respiration movements, the ear can take no note of intimate changes even though these should imply actual destruction of the organ. Between the consolidation that is recoverable and the consolidation that necessarily implies death, there may be no acoustic difference whatever. Thus the progress of the local changes which accompany pneumonia is not to be followed at every successive step; at some places its course is obscure, and at some we, for a time, lose trace of it altogether. It is this imperfection in our means of

Whatever may be said for or against the occurrence of a stage of the disease anterior to that of pulmonary engorgement, no one would expect that the reality of such a condition could be established anatomically. In the highly improbable event of death at that early period it would remain for ever uncertain whether or not pneumonia had actually existed. Anatomically, therefore, the affection first becomes recognisable in hyperæmia of the lung. Yet even here, as has been repeatedly said, hyperæmia does not of itself indicate, it need not even suggest, pneumonia. Active pulmonary congestion owning a quite different origin may put on a precisely similar appearance, only to be distinguished from pneumonic engorgement by the help of collateral circumstances.

The earliest anatomical sign.

Nevertheless this very hyperæmia, recognisable or not, is the immediate preliminary to the great event of pneumonia, the escape of blood plasma from the capillary vessels, and its deposition in the pulmonary alveoli. A portion of lung so affected is obviously blood-laden, redder, denser, and less yielding than the rest. It is not airless, but the creaking under the finger differs from that in health. Such air as it contains, though not easily expelled by squeezing, may be pinched hither and thither. Placed in water the piece neither floats on the surface nor sinks to the bottom, but swims higher or lower as the air remaining is more or less. From a cut section of such lung a thin blood-stained fluid can be pressed, more or less frothy. Looking closer it

Engorgement.

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observation that gives rise, as we shall see, to some of the perplexed questions of the subject. How far may local changes proceed with possibility of recovery? What specimens of ours represent cases that must inevitably have died, and what those that might possibly have recovered? and so forth. It is in view both of the imperfection and the unequalness of our knowledge in regard to this branch of the subject, and the impossibility at certain points of connecting the so-called 'physical signs' with the actual physical state, that the morbid anatomy of pneumonia is to be described.

is possible to discover, with the help of a simple lens, both between the lobules and immediately under the pleura, little points of extravasated blood. In thin section under a low power of the microscope, as the 2-inch, the capillary vessels may be seen 'turgid and tortuous,' encroaching upon the alveolar spaces. The mucous membrane of such small bronchules as the specimen contains may or may not exhibit increased vascularity. The air sacs themselves at this period are unequally laden with watery fluid. In a thin section comprehending a number of contiguous sacs, this will appear in the distension of some and the partial or even entire collapse of others, a condition which disappears at a later stage. It may be added that washing fails to remove the heightened colour of this hyperæmia, a point in which it is said to differ from passive congestion.

Red hepatisation,

Succeeding to this appearance and often seen along with it is consolidation, 'red hepatisation' as Laennec well called it, although, speaking strictly, its colour is not red but reddish brown. The tint is not uniform but indistinctly mottled, with here and there lighter shades suggestive of grey. The lung now for a whole lobe or the greater part of one has the consistence and something of the appearance of liver. Its spongy texture, with the physical characters that depend thereupon, is entirely lost. It sinks in water. Forcible pressure between the fingers does not compress but breaks it. The resistance it offers to the knife is like that of the liver itself, but the cut surface is not smooth but finely granular, consisting of a number of minute elevations like the outside of a nutmeg. This peculiarity of structure is better seen when the knife incision is prolonged by tearing. Sometimes, by squeezing or scraping with a scalpel, little plugs or moulds of soft yet solid-shaped material may be detached, similar both in size and shape to these granulations. This same material is in some instances continuous with a fibrinous deposit extending to the larger bronchi.

its signification;

Now this so-called second stage of pneumonia consists essentially in the deposit of blood fibrin within the alveoli and infundibula. Observe that in this process, whereby the lung is rendered solid and impermeable by air, there is no destruction or even change of the proper lung tissue. The alveolar walls remain as in health, only thickened, it may be, temporarily, owing to the dilatation of their capillaries; but they enclose now a plastic material moulded into the shape of the air-sacs, and embedding, as we shall see, certain histological elements.[1]

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[1] The actual *seat* of the inflammation products being no longer matter of dispute, the question turns next upon the *source* of this deposit. Whether are the bronchial or the pulmonary capillaries concerned, or are they both concerned alike? On the one side Dr. Morehead ('Diseases of India,' vol. ii. p. 312) regarding the matter as from its nature not capable of demonstration, argues that the bronchial capillaries are the proper nutrient vessels of the lung, while the pulmonary have a special destiny and purpose in connection, not with nutrition, but with the aeration of the blood. In inflammation of the lung, therefore, it will be, he contends, the former vessels that are first and immediately affected, as taking the chief part in the vital processes of nutrition of the cell walls. The pulmonary capillaries, he maintains, are affected only secondarily, although their 'deranged action has much to do with the pathology of pneumonia.'

On the other hand Dr. Waters ('Diseases of the Chest,' p. 27), demurring to the view that the point is to be settled by analogy and hypothesis, undertakes to demonstrate that it is the pulmonary and not the bronchial capillaries which are at first involved. 'In some cases of pneumonic inflammation,' says he, 'there is no reddening of the mucous membrane of the finest bronchial tubes'—no evidence, that is to say, of inflammatory action at the point to which the distribution of the bronchial arteries extends, and where it comes to an end. If, therefore, he argues, 'the air sacs alone are the seat of the exudation, it is obvious that the latter must be poured out from their walls.' Now these walls contain no other vessels than those derived from the pulmonary artery. Therefore the exudation must be poured out by them.

The question is hardly of practical moment. In the usual course of events, probably both sets of capillaries are involved. Yet I cannot but believe that Dr. Waters is in the right, and that his the accompanying pleurisy,

The pleural surface of such a lung is not less characteristic than its substance. Here the brown red of the consolidated organ is in part (or it may be altogether) concealed by a layer of soft fibrin unevenly spread, and apt to be disposed in patches and loose shreds. These latter are the representatives of bands adherent in life to the costal pleura, and which on the removal of the lung have parted from their attachment. The emptied cavity of the chest is lined or smeared with the like material, and it is often easy to make out in places by the roughened and jagged surface the precise points at which the lung had by its agency become adherent to the chest walls.

an infallible evidence of pneumonia.

This transformation of the light, spongy lung tissue into a heavy,[1] solid, and perfectly airless structure, over whose surface a grey or ash-coloured layer of lymph is spread of varying thickness and consistence, is perfectly characteristic of true pneumonia. With the feeblest description it can never be mistaken or overlooked. True, the degree of solidity, the amount of blood-stained fluid which exudes on cutting, the depth and uniformity of the colouring, will vary as well with the stages of the process as with the period that has elapsed since death. Such modifications, however, will never so far transform the early hepatised lung of acute pneumonia as to disguise for an instant the disease of which it is the evidence.

Red hepatisation passing into grey.

As hepatisation proceeds the lung begins to lose colour and soon becomes distinctly mottled with grey, and, as it pales (I am speaking necessarily of the fatal cases), it becomes less dense, and in isolated spots, which are thus in advance of the rest, so far softened and so colourless as to

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observation that inflammation does not necessarily extend to the bronchules is strictly accurate.

[1] See, for example, Cases 11, 12, 16 of Class IV., Appendix D. where the weight of the affected lung is three pounds and more.

answer to the description of 'grey pneumonia.' It is only rarely that a hepatised lung is met with in the post-mortem room that does not exhibit on close inspection certain spots here and there which are thus beginning to grow pale and soften. Yet from the nature of the process we may fairly assume (which indeed is not absolutely without proof) that this softening is but the sequel of solidification, representing only certain lobules or districts of lung further advanced than the rest.

Different rates of progress in contiguous parts.

Thus, from the evidence of morbid anatomy no less than from the physical signs during life, it seems probable that the course of pneumonia does not consist in the simultaneous and uniform transformation of a large area of lung from one state to another. It is rather a process advancing unequally in different places. Sometimes there is more and sometimes less uniformity, yet almost always (so far as post-mortem observation teaches), pneumonia, considered as a local process, is to be seen at various stages of progress in one and the same lung. The changes which are correctly described as successive for each individual lobule are apt also to be synchronous as regards the entire portion of lung concerned.

Changes subsequent to death.

Red hepatisation, it has been suggested, becomes rapidly altered by the fact of death, and does not exist in the living body as we see and describe it. In lungs so affected examined early, blood exudes plentifully; in those we are in the habit of inspecting at hospitals after an interval of from 12 to 20 hours, a thin reddish fluid only remains. The inference is that the anæmic condition is due to post-mortem change, and that the elasticity of the exudation, (which in life the force of the heart's contractions was able to overcome), suffices after death to drive out the blood.

Nature of the process.

The pneumonic process, therefore, regarded from the anatomical point of view, would seem to be both an exudation and a hæmorrhage. With certain preliminaries as to which anatomical evidence does not permit us to speak precisely, its first recognisable phenomenon is the pouring out over a large tract of lung of the blood plasma, while at numerous points, as by the force of the inundation, the capillary vessels are visibly rent and the blood itself escapes.

Minute histology of these changes.

But it is not this only, or even chiefly, that we meet with in microscopic specimens of the lungs of those who have perished by pneumonia. The invasion of the alveoli necessarily induces further changes, and it is not until these have occurred that the patient succumbs and his organs are open to inspection. We find then scattered through a finely fibrillated coagulum which occupies the alveolus, certain corpuscular elements various in various examples of the disease, and only to be identified at an early stage of their life. The natural course of events appears to be for this fibrillated material to become more and more beset with these corpuscular elements, and for its delicate network gradually to fade away before them. Hence, with our late opportunity of inspection, we get to regard this latter condition—the occupation, namely, of the alveolus solely by corpuscular elements with no demonstrable bond of union—as specially characteristic of the hepatised lung. There is reason to believe, on the contrary, that the fibrinous interlacement is constantly present in every instance of true pneumonia, and that with its disappearance we lose (so far as any particular microscopic specimen is concerned) the means of discriminating the disease with certainty.

Appearances commonly observed in consolidated lung.

It must be admitted, at least, that the formed elements recognisable in consolidated lung offer considerable variety, and that it is difficult, or rather impossible, to connect these varieties with corresponding clinical groups. In hepatised lung generally, whether of pneumonia or not (in the sense in which we are now using the term), there are to be seen, along with coloured blood-discs which are never absent and sometimes abound, multitudes of cell forms, some of them, as is asserted, the result of proliferation and subsequent transformation on the part of the natural epithelium of the alveoli, others migrated leucocytes, the representatives of the white corpuscles.[1] Of these latter some are indistinguishable from those to be seen in the living blood, and some so transformed that their nature and origin is rather matter of conjecture.

The further progress of hepatisation.

Now as hepatisation proceeds the variously-formed cells regarded as epithelial undergo fatty degeneration. Their place becomes occupied by coarse granule cells, while by the setting free of their nuclei and the mingling of these with the corpuscular elements, the entire contents of the alveolus begin to assume one form. It becomes crowded, that is to say, with little roundish or ovoid granular cells, with and without nuclei. These, when in the thin section they are accidentally dislodged, exhibit considerable coherence. Sometimes they hold on by delicate strands to the alveolar wall, and sometimes between contiguous groups of them tiny meshes are to be made out, suggestive still of a finely fibrillated network encircling the whole.

Such, I say, is the appearance which the pathologist associates with pneumonia, inasmuch as, in the nature of things, it is not at the very commencement of hepatisation that the lung is likely to come into his possession. It is to be remembered, however, that this uniform massing of pus-like cells denotes in fact an advanced stage of the process. The exudation has then reached that period in its history which immediately precedes either its disappearance and recovery of the lung, or else the participation of the proper pulmonary structure in a process which thus first becomes directly destructive.

Process of recovery or resolution.

Already, in some specimens, the former and favourable change may be seen, as it were, in actual progress. The new material is lying loosely within the alveolus, retaining but a feeble hold upon its wall. Between the latter and the shrunken mass of agglutinated cells a free space can be made out, or, sometimes, the separation is incomplete and the morbid product may be seen moored to its place by some still adhering fibrillæ of that fine network which holds together its separate elements. In other cases the little mass, detached wholly from the alveolar wall and destitute of fibrillæ, is itself undergoing a further transformation in becoming converted, from circumference to centre, into a finely granular material.[1]

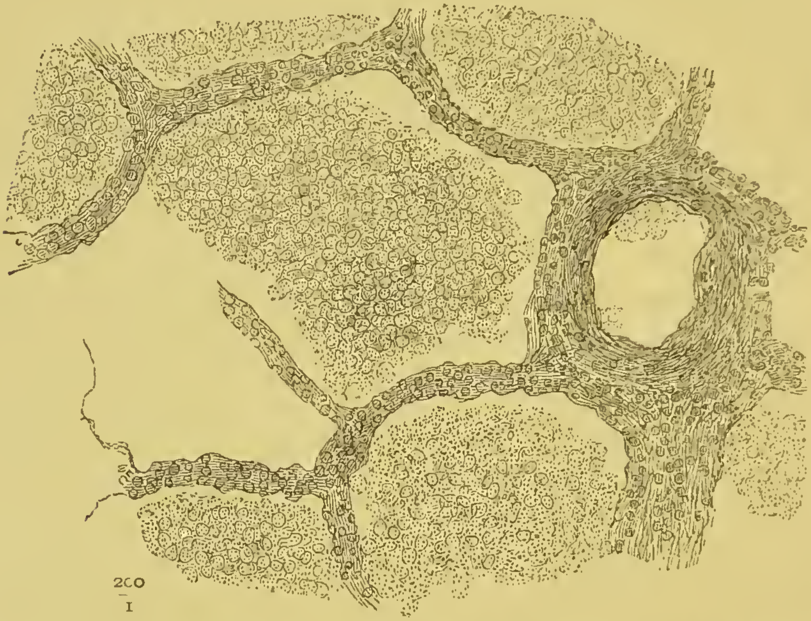

Fig. 1.—Hepatised lung in process of resolution. From a patient with pneumonia dying when the physical signs of resolution had been established. The corpuscular elements lying loosely within the alveoli have undergone a granular conversion at the circumference.

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[1] The mere fact of the exudation lying loosely in its place is not to be taken as evidence of commencing resolution. The appearance may be artificial and occur during the preparation of the specimen, or it may indicate that stage of conversion presently to be described (see p. 110).

From this disposition of the alveolar contents, it is thus sometimes evident in examples of pneumonia which nevertheless die, that in certain districts of the lung this foreign occupation was about to cease, and that at the moment of general surrender a particular spot was on the point of being relieved.

The histological condition at the period of resolution is probably different in different cases.

It is obvious that we can know but little from direct observation of the successive steps in the progress towards recovery of an organ which is out of sight and reach. The destruction of the patient at such a period, even by some cause remote from the lung, would modify, if it did not arrest, a process of restitution like this, which demands for its due fulfilment an unimpaired vitality. It is commonly taught that the delivery of the lung and reversion to health is never accomplished save by a purulent conversion of the alveolar contents. The assertion cannot be established upon clinical evidence, and we must hesitate to accept it on that of fatal cases. These, since they never reach the particular stage in question, are not likely to exhibit its peculiar phenomena. It seems not improbable that in the resolution stage of ordinary pneumonia the corpuscular and epithelial contents of the air vesicles (unrecognisable as yet as pus corpuscles) undergo very early granular degeneration and liquefaction, and are so disposed of.[1] In instances of greater severity and longer duration the transformation into pus may be nearly or quite completed.

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[1] 'Pus cells,' writes Dr. Wilson Fox, 'are produced throughout the whole pneumonic process.' Dr. Hughes Bennett believed that a molecular exudation into the air vesicles becomes converted by 'molecular coalescence' into pus cells, and that these subsequently undergo degeneration. Since this was written, however, many observations, and especially those of Cohnheim, have thrown doubt upon the reality of this antecedent molecular exudation. The appearances figured by Dr. Bennett are nevertheless unquestionably faithful, but the aggregated molecules which he represents are not making into pus cells, but *are these bodies breaking down*; the molecular material is but the final disruption of an exudation which at the first was formed and corpuscular.' (See Dr. Bennett's 'Restorative Treatment of Pneumonia,' pp. 49 and 50.)

While no anatomical description can include the resolution stages of pneumonia or the successive steps by which the lung recovers itself, as little can we determine by direct evidence how far the destructive process may proceed with possibility of ultimate recovery. It is only with the patient's death, and the inspection of his diseased organ, that we can trace the course of destruction through its successive steps. It is only after reviewing a large number of such observations that we can venture to estimate the extent to which lung disorganisation from this particular cause can possibly be carried before the process is inevitably arrested by death.

CHAPTER VIII.

MORBID ANATOMY OF PNEUMONIA—*continued.*

Purulent conversion—Destruction of the lung tissue—Concurring changes on the part of the pleura—Various rates of progress of the morbid process—Condition of other organs—Summary of morbid phenomena.

The process of disintegration.

As has been said, the change from red to grey hepatisation is not abrupt. Already, in most of our specimens of the first condition, an obscure mottling of the surface betokens the commencement of the second. In many this mottling has become so distinct that a section will resemble nearly some kinds of red granite. As the areas of greyness extend so does the organ as a whole get less dense, while the grey patches themselves soon become obviously softened, a grey opaque fluid exuding from their section as the knife-edge is passed over it. Soon the whole of the affected lung shares the same condition, the mottled appearance fades in its turn, and with it all trace of the rich brown-red of early hepatisation. The surface is now of a uniform or nearly uniform grey, pale, bloodless, and in parts almost pulpy. Its section no longer exhibits a granular surface, and in the attempt to tear it the structure wholly breaks down into putrilage. It melts before the finger as does the wet sand of a receding tide.

Yet even at this stage it is often possible to convince oneself that the proper structure of the lung still remains, although now textural changes have set in to threaten, if they do not imply the ultimate ruin of, the part.

Microscopic appearance of purulent infiltrations.

In effect, what this grey or yellow hepatisation signifies, and what in its various stages it exhibits to the microscope, is the gradual liquefaction of the alveolar contents, with ulcerative destruction of the intervening walls; and finally, conversion, alike of the structural and of the morbid elements, into large districts of pus cells in which the proper tissue of the lung is represented only by scattered fragments. One cannot, of course, follow during life the successive steps by which this change is wrought, yet it is easy to recognise afterwards its several stages and to fix their date in relation to each other. The manner of progress is probably of this kind. First, the masses of pyoid cells occupying the separate alveoli begin to dwindle so as no longer to fill up the entire space. They become spotted with black pigment. The delicate network of fibrillæ which at first connected them has now disappeared; in its place is a thin fluid in which each little

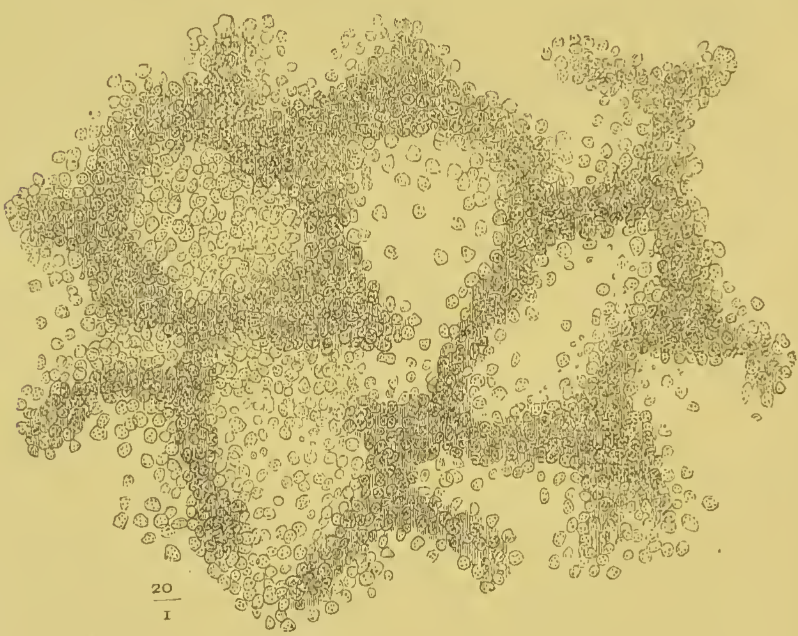

Fig. 2.—Pneumonia in the grey and softening stage. The alveolar walls exhibiting corpuscles similar to those they enclose, and in places showing signs of breaking down.

cell mass is immersed. And now, for the first time, there appears in the meshes of the capillary vessels which constitute, or rather help to constitute, the alveolar walls, multitudes of young cells which are often indistinguishable from those which they surround.

Implication of the alveolar walls.

This is a condition which, if it could be seen in the living body, would by no means decide the ultimate fate of the lung. On the one hand, by the melting away of the exudation (evidenced by the dwindled cell contents, the accumulating fluid, and the disappearance of meshes of fibrin), its final expulsion is facilitated ; on the other, the corpuscular infiltration of the alveolar walls implies a danger to the capillary circulation, and so to the vitality of the part.

Cell proliferation.

But there happens often at this period, (either with or without this alveolar-wall growth) between the dwindling cell mass and the wall of the alveolus, a proliferative process on the part of the latter, a development of epithelial-like cells, a catarrh, it may be, excited by the presence or the character of the exudation. By this intervention, as some maintain, both the escape of the exudation is mechanically prevented, and the outward pressure from the ever-accumulating contents of the alveolus is sufficient to arrest the capillary circulation. This at least seems certain, that with the continuance of this proliferation the rapidly-formed elements assume more and more the purulent character, that they mingle with the pyoid cells which have occupied the alveoli from an early period, and soon become indistinguishable from these. Meanwhile the alveolar walls, partly from a corpuscular growth of their own and partly from rupture under distension, become indistinct and broken. Yet the disorganisation thus accomplished—true to the character which distinguishes these morbid changes throughout—is not uniformly exhibited. In one place the proper lung tissue will remain almost entire, in another no more will be seen than a crowding together of pus cells.

Transformation and rupture of alveoli.

To these transitions in the intimate structure of the lung there corresponds, as we have seen, a gross and palpable change of consistence. The organ, through various shades of grey, becomes eventually yellowish, and obviously pus-laden, and although, even at this ultimate stage, its delicate framework is still preserved for the most part, the whole is soft and miry, and ready to melt under pressure.

The inflammatory products of the pleura correspond with those within the lung,

While such phenomena are being exhibited on the part of the lung, its pleural investment is not quiescent. The fibrinous exudation upon the pleura undergoes, as I believe, a succession of changes, in strict conformity as to time and character with those just described for the lung itself. Thus, when pneumonia is early resolved, and the fibrinous exudation into its air-sacs undergoes early transmutation, the pleurisy is of the plastic kind, and ends often in adhesion; but where the lung exudation suffers a further degradation, and is at length converted into pus, as in most of the fatal cases, there, too, the pleura sympathises *pari passu*; from fibrinous it becomes purulent, and when inspected along with the lung is creamy and semi-fluid, indistinguishable, as regards its histological elements, from the contents of the alveoli.

unless adhesion or old membrane interfere.

This sympathy on the part of the pleura is sometimes interfered with, owing to the presence of false membrane, the result of a previous attack of simple pleurisy; a tough layer of fibrous material of varying thickness will so embrace the lung in such a case that the natural development and transformation of the proper pneumonic pleurisy will be impossible. Old pleuritic adhesion would have a similar effect. Or, again, the pleurisy which accompanies the pneumonia may at once give rise to adhesion, and so end, while the exudation within the lung pursues its course alone. Nevertheless, both within and around the lung a single morbid[1] process is in operation, which such circumstances of situation conspire to modify.

The disease varies with the rate of progress of its anatomical stages.

The varieties of pneumonia, anatomically considered, have reference, as we have seen, to the area over which the disease extends, and the manner of its spreading from point to point. The rate of progress, though beyond the recognition of anatomy, is another feature of difference. In one instance the affection will reach its resolution stage when but a small portion of the affected lung has become solid; in another, purulent conversion will be so rapid and so uniform that one is entitled to doubt whether the lung has reached that condition by the usual steps.[2] Such variations, however, do not call for notice in this place, inasmuch as they concern chiefly the clinical history of the disease.

Corresponding changes in other organs.

I have said that the morbid phenomena of pneumonia are not confined to the lungs. This is sufficiently shown in its general history. It is less easy to exhibit these associated changes anatomically. Death may efface or exaggerate them. A patient so far outlives the acute typical stage of his disorder as to lose all trace of those secondary lesions which depend immediately upon it. Or, contrariwise, his death may be so sudden that it becomes difficult to separate what is due to the disease from what is due to sudden death everywhere. We know nevertheless that the condition which accompanies pneumonia is one of intense hyperæmia of

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[1] Dr. Addison, it will be remembered, in discussing thirty years ago with his wonted sagacity the pathology of pneumonia, draws a comparison between the air cells and the serous membranes, likening the effusion of serum into these to the serous effusions into the pleura and peritoneum. See his paper on 'Pneumonia' in 'Guy's Hospital Reports,' 1843.

[2] See, as examples, Cases 18 and 19 of Class V., and 2 of Class II., Appendix D. It was held by Dr. Hodgkin that purulent infiltration might occur without preceding hepatisation. See his 'Lectures on Pathology,' p. 98.

State of the heart-cavities.

the internal organs generally; that this is conspicuous almost always in the kidneys, and often in the liver.[1] The heart suffers by distension, especially of its right cavities, insomuch that death may occur, as we have seen, directly from its failure. The blood exhibits a tendency to deposit fibrinous clots, the entanglement of which in the pulmonary vessels is one of the modes of sudden death in pneumonia. Almost invariably, when the inspection is early enough, both ventricles, but especially the right, will be found to contain firm and often partially decolourised clot. This sometimes so moulds itself into the network of the columnæ carneæ, and reaches so far into the large vessels, that in spite of its firmness it is difficult to remove it entire. It may be added, that in fatal cases the implication of the pericardium is so common that this too may be regarded as among the pathological associations of the disease, but of this we shall have occasion to speak presently.

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[1] Dr. Bristowe ('Path. Trans.,' vol. viii. p. 66) has remarked upon the fact (which he supports by reference to post-mortem records) that acute dysenteric ulceration is not an unfrequent accompaniment of acute pneumonia. He adduces this circumstance in illustration of the principle that 'when acute inflammations are the result of the operation of general causes the local manifestations are by no means necessarily confined to one organ' The principle thus enunciated may well be conceded, and at certain epochs (as at the time to which Dr. Bristowe refers) even illustrated in this particular way. But it is less than certain, I think, that the statement can be maintained as a general one, true of pneumonia at all times. We are told upon the high authority of Dr. Morehead ('Diseases of India,' i. 5 4) that dysentery has for its immediate exciting cause not malaria but cold, and we know that cold is also not seldom the immediate exciting cause of pneumonia. It might, therefore, provoke both at once. Nevertheless the actual alliance of these two diseases is, in ordinary times, a rare event, because the causes predisposing are different. It is not cold *alone* that can produce either the one or the other, and the body predisposed towards the one seems, as a rule, on that account indisposed towards the other. Dr. Bristowe's observations probably refer to a period at which the epidemic constitution was in this respect peculiar. The early history of pneumonia seems to show that the concurrence of the two diseases has happened before.

Summary of events.

It will not escape notice that there are certain points in the foregoing account which need further elucidation. Here and there it is rather by inference and hypothesis that we connect the several anatomical phases in the disease, so as to frame a consecutive history. Yet the main characters of the pneumonic lung, and its successive transitions, may be thus summarily stated :—

1. The chief histological events of pneumonia would seem to be these :—The pouring out of an exudation into the air vesicles through a large district of lung, yet variously in various parts of it; first, of a mere watery fluid, presently of the fluid plasma of the blood, with its formed elements, and here and there, at separate little points, by actual rupture, of undisguised blood. Along with this exudation or hæmorrhage a corpuscular generation takes place, representative partly of migrated white corpuscles, partly of epithelial overgrowth. So soon as the exudation begins to solidify, these products become apparent, entangled in the meshes of it. Next, this intervening network itself disappears, and the closely-crowded corpuscles are held together by their own stickiness. They, too, are undergoing change, some of them resolving into fat, and represented by coarse granules. Then comes a loosening of this corpusculated mass, its granular transformation, liquefaction, and final removal mainly by absorption. Catarrhal proliferation is a variable incident of this process, sometimes aiding, sometimes retarding, and sometimes arresting it.

In the failure of this process of removal the proper lung structure becomes implicated secondarily. The corpusculation, which was at first wholly intra-alveolar, begins to infiltrate the alveolar wall, and both here and there to assume the character of pus. The boundaries of the several air-sacs are thus rendered indistinct owing to this common implication, and soon are only to be made out by faint cloudy paths. At length these partitions breaking down at many points and coalescing with the alveolar contents, there remains no more than the débris of lung structure, recognisable chiefly by scattered pigment.

2. The theatre of the pneumonic process, so far as the lung is concerned, is not the lung texture, but the lung spaces. The implication of the alveolar walls is secondary and accidental. In the ordinary non-fatal career of the disease it is probable that these undergo no structural change, as it is certain that they sustain no permanent injury from this temporary lodgment of a morbid product.

3. True pneumonia cannot be certainly recognised at the post-mortem table until the stage of consolidation is reached. It is then highly characteristic by its colour, consistence, and pleuritic envelopment, insomuch that a naked-eye inspection will often inform us better of the nature of the disease than will a minute examination of individual lobules.

4. Although we speak compendiously of the several stages of pneumonia, ascribing to each certain special features, yet, in fact, the disease exhibits throughout one constant histological element. Its changes from red to grey, and from grey to yellow, are due less to a change in the nature than in the gross amount of the exudation. By the accumulation of this material the blood, which in the beginning was distributed to the lung in excess, is, so to speak, squeezed out. Hyperæmia ends in anæmia. It seems probable that in life the power of the circulation to overcome this resistance is maintained up to a very late period, and that our terms, so far as they bear upon colour, are less applicable to the living than to the dead lung. Thus mere colour is of itself a fallacious guide, and must always be regarded in connection with the time and the mode of death.

5. Neither abscess nor gangrene is among the consequences of pneumonia.[1] Where the disease is not fatal it is the rule for the implicated lung to be restored whole as the other. That there are instances of the contrary has been already implied. Tissue degradation, progressive or stationary, may seem to take its start from the acute attack. Not only by corpuscular infiltration may a portion of lung be permanently damaged, but further, fibroid overgrowth may occasion thickening of the alveolar walls with corresponding diminution of the cell spaces. And these two processes are apt to blend and to replace each other. It is of doubtful propriety to speak of such conditions as 'chronic pneumonia.' Even admitting them to be the direct results of the pneumonic process, they differ essentially from that process itself, and are only recognisable upon its departure. The subject, however (which involves the connection between pneumonia and phthisis), will be best considered apart.

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[1] See Appendix F, Pulmonary Gangrene.

CHAPTER IX.

FORMS OF LUNG CONSOLIDATION DISTINGUISHABLE FROM PNEUMONIA.

Alveolar catarrh simulating pneumonia—Fundamental difference between the two—Alveolar catarrh in young children—Its origin from collapse—Varieties—Relation to true pneumonia—Association with certain diseases and diathesis—Lobular collapse and its results in old age.

Certain forms of alveolar catarrh may simulate pneumonia.

IN the previous chapters I have endeavoured to describe the usual aspect and phenomena of pneumonia as well as to point out certain forms of the disease which, while they diverge from the typical pattern, are yet admitted into the same category with it in virtue of a real kindred which circumstances have conspired to obscure. We have to consider now examples on the other side, where an outward similarity coincides with an essential unlikeness. Presently, by a comparison of these two classes of cases, it may be possible to strike the actual line which separates pneumonia from its counterfeits.

Leaving the dead for a while, let us return to the living subject. Take such a case as the following :—

Case of acute alveolar catarrh.

George S., a labouring man, aged 33, of somewhat intemperate habits, was admitted into the Westminster Hospital under my care on December 16, 1873, with the following history. For some winters past he has suffered from bronchial catarrh, but only occasionally has the affection compelled him by its severity to leave work. While so suffering, yet able to get about, he was seized on the 11th (5 days since) with cold chills, dyspnœa, and pain in the left side with increase of cough. After three days' endurance of these symptoms he got in addition hæmoptysis, coughing up, as he thinks, more than a pint of blood. When the pure hæmorrhage ceased the sputa became rust-coloured, that is to say, expectoration similar in consistence to that of the chronic catarrh was now uniformly stained with blood. It so continued up to the time of his admission.

When seen on the 16th his face was flushed and anxious, he lay on his back, not raised, the pulse beating about 120, temperature 102·8°. Sonorous râles were to be heard over the whole chest, and the lower half of the left lung yielded a true though somewhat coarse crepitus. There was dulness over this spot which, with defect in the vocal vibration and altered character of the voice sound, sufficiently indicated fluid in that pleura, not in large quantity nor displacing the heart. The urine, with excess of urea, still contained chlorides and showed a trace of albumen.

Without reporting this case in detail, the following were its main features. The sputa soon became very abundant, unaerated, rust-coloured in part, and in part uncoloured or greyish ; the auscultatory sounds continued without alteration, no bronchial breathing being audible over either side. The body heat rose to 103°, its highest point, on the second day from admission, being the seventh day from the initial attack. The following day, with temperature down to 101·3°, the man's general condition was not better, and he had to be freely stimulated. On the 20th, with temperature still falling, he had a soft, very compressible pulse of 120, and a drying tongue, and had been kept from sleep by paroxysmal cough. There was some extension of the posterior dulness, still universal bronchial râles, with coarse crepitation of base of left lung, and still an absence of bronchial breathing. The expectoration was now of a viscid uncoloured mucus. The best feature of his case—which was now a very grave one—was his acuteness of perception and attention to his own distress.

The patient continued in a critical condition for some weeks, pyrexia persisting and the temperature varying from 99·7° to 102°. The respiration gradually became whiffing as the fluid increased in the left chest. The sputum, after it had finally ceased to be blood-stained, underwent several changes of colour and was for a time of a distinct chocolate. He had several 'critical' amendments which were only temporary, especially on December 27 and January 2, 16 and 21 days respectively from the commencement of the illness. The first of these was marked by a distinct change for the better in the man's aspect and manner. He had also a rigor followed by sweating and subsequent general amendment on January 9. On the whole, however, his recovery was very gradual, and cannot be said to have fairly commenced till he had been ill nearly a month. The local signs receded by equally slow degrees, crepitus being audible throughout. The man wasted in a marked manner during the illness, and when finally made an out-patient, after six weeks' confinement to bed, had still the bronchial rattles in his chest, and expectorated a thick yellow mucus.

Its points of difference clinically.

We have here an assemblage of clinical phenomena closely resembling, even to aspect and manner, those that have been described as proper to pneumonia: a sudden seizure with distinct rigor, pyrexia and dyspnœa, veritable rust-coloured sputa, crepitation over one lung, and more than once critical phenomena in sudden remission of pyrexia, profuse sweating, &c. Nevertheless, no one of these symptoms can endure the test of close interrogation. Take for example the sputum. In addition to somewhat profuse hæmoptysis, this becomes coloured for a while with blood, and that repeatedly. On the hypothesis of pneumonia, therefore, the course of this one symptom would imply not one but a succession of attacks. Again, the affection drags on indefinitely for weeks with little variation, with an irregularly oscillating temperature, with no extension or progress of any kind on the part of the physical signs, and no indication at any time of the lung becoming solid. The crepitation which marked the onset of the affection persists, and all the change observed is outside the lung, consisting in a gradual increase at first, and a gradual decrease at last, of fluid within the pleural cavity. This is no mere modification of pneumonia, both the elements and the agency are different.

Cases like these, of which in their general progress and prolonged yet imperfect recovery the foregoing example may stand in some sort as the pattern (though no single instance can figure the condition fully), may be contrasted with pneumonia clinically thus :—

While rarely an affection of youth or early manhood, it is associated especially with the ultimate stage of certain infantile and senile disorders.

Although in its actual attack it is apt to be sudden, it does not occur to the healthy and robust, but assails those already weakened by chronic illness, and particularly where bronchial catarrh has been a prominent feature of such illness.

The march of the affection is not by regular steps involving a succession of physical changes on the part of the lung, and ending abruptly at a certain stage.[1] It is a course of ups and downs of indeterminate duration. At every point in its history there is the liability to a recurrence of the earlier symptoms, and when it finally departs there still remains the probability of its return, which every successive attack renders greater.

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[1] The presence throughout in normal quantity of the urinary chlorides is a point that might also be insisted on, but this is not invariable, nor, as we shall see, do these chlorides always disappear in true pneumonia.

Opportunity seldom occurs of inspecting the lungs of these patients in what may be called the acute stage of their disorder. It is only after surviving several attacks of the kind that the secondary changes thereby induced, both in the lungs and in other organs, combined often with the feebleness of age, conspire to destroy life. We see then a condition essentially differing from pneumonia in a subject with whom the occurrence of that disease, if not impossible, is at all events extremely rare.

Points of difference anatomically, in the lungs,

The anatomical character of this condition may be described as follows :[1]—In colour the lung is a mottled grey or slate colour, neither red nor reddish brown. It is solid, but without the firm and uniform solidity implied by the term hepatisation. Consolidation is unequal and incomplete ; by handling and squeezing it we may often get to suspect what further inspection afterwards reveals. In the section of such a lung it exhibits separate islets of consolidation. Here and there, in narrow tracts or over broad areas, portions of still crepitant lung may be mapped out. Add to this that there is intense and extensive congestion of the bronchial mucous membrane, while the fellow lung, although falling short of actual solidity, is yet hyperæmic, and, as well by congestion as by the abundance and character of the secretion within the tubes, gives evidence of general bronchitis.

pleura,

As regards the pleura recent pleurisy is exceptional, while it is quite common to find serous effusion. The heart will probably have undergone that change which is both the cause and the effect of prolonged pulmonary congestion, the change, that is, from an overstrain which has elicited no response. The right ventricle is dilated and its wall thin, and fat is encroaching from the apex. The muscular fasciculi themselves sometimes exhibit fatty or granular degeneration. These conditions may exist (as in the particular case from which I am drawing) where the kidneys and liver are practically healthy.

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[1] I am taking for my model in this description a particular example where a seaman of about 40, after a succession of such attacks as the one just described, died at last in the Westminster Hospital, Nov. 25, 1874.

and heart.

Whatever name may be given to this unequal consolidation, it is obviously incorrect to describe it as lobular. Although the denser portions are scattered, and even isolated, these do not coincide with individual lobules. The alveoli suffer in groups: the anatomical divisions are disregarded.

Microscopic appearance.

If a thin section of such a lung as has been described be placed under the microscope, it may or may not exhibit the appearance which histologists recognise as catarrhal pneumonia. It is often, I think it is almost always, possible to find in such a lung groups of alveoli distended with corpuscular material, indistinguishable from pneumonia at its later stage. But along with these, and in strict correspondence with the unequal density of lung which mere handling discovers, there are tracts of lung whose alveoli, unequally filled, exhibit distinctly large epithelial cells budding from their walls. It is, in fact, by the different rates of progress of this proliferation in neighbouring alveoli, distending some at the expense of the rest, and by the mode in which these catarrhal products are thus germinated from the alveolar walls, that this catarrhal condition differs widely from that uniform packing of alveoli with corpuscular contents which nevertheless may often be seen along with it.[1]

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[1] The condition alluded to is intimately associated with collapse, and will be illustrated further on in that connection. See p. 129.

Alveolar catarrh in young children induced by neglect and by certain diseases.

It is in young children most markedly that we see, or more often that, without seeing or suspecting it in life, we find after death, a form of alveolar catarrh which is in many respects similar to that just described. This so-called lobular pneumonia is a secondary affection connected with a particular 'diathesis,' and associated particularly with at least two infantile diseases; it is induced, in its favourite subjects, by bad nutrition and bad dwelling, insomuch that it would be possible, no doubt, so to arrange circumstances as even to obtain it at will. It conduces in a latent and insidious manner to permanent and irremediable changes in the structure of the lung.

Common example.

Here is a weakly child—to describe in general terms what has happened repeatedly—recovering from some acute disorder which has bronchitis as a common sequel. Continuing to suffer in that way the child wastes, has nightly fever, with hurried wheezy respiration, and, perhaps, occasional fits of serious dyspnœa. Such symptoms, now better and now worse, and represented by a very irregular temperature chart, will go on for an indefinite time. The affection, in the observation of the physician, makes no progress of any kind. Physical examination often repeated reveals the same thing from day to day; various sounds of increased secretion in the larger tubes heard over both lungs, a little finer or a little coarser here or there, but at no time approaching crepitation, or with any sign of consolidation. Such a patient, it is true, will sometimes, along with sudden dyspnœa, develop such signs all at once at a particular patch or patches of lung. More commonly, however, events are gradual. There is an intensifying of existing symptoms rather than an importation of new ones. The evening fever is continued into the day, the dyspnœa is aggravated into paroxysms of extreme breathlessness, while the ear, watchful and expectant, fails to catch any sound within the chest to give notice of what is nevertheless suspected.[1]

Implication of the alveoli.

When these infants recover we call the condition bronchitis, for there has been no physical evidence of more. When they die it has to be called lobular pneumonia, for it is found that, in addition to bronchitis, certain lobules are airless, discoloured, and condensed. The inflammation, we then say, has spread itself to the vesicular structure of the lung, 'pneumonia has insidiously ingrafted itself upon bronchitis.'[2]

Nevertheless the minute examination of such lungs does, in fact, bear out what the living symptoms indicated. The children do in reality die, as they seem to do, of acute bronchitis, and these condensed spots have a significance as illustrating one particular feature apt to attend bronchitis in infancy. These altered lobules or groups of alveoli, in the case of children that survive the acute stage of their disorder, have a future history to fulfil; yet, so far as the immediate illness goes, they are but an incident of the bronchitis, and both in inception and progress are too often hid from observation.

Mechanism of lobular collapse.

Most readers are probably familiar with the investigations of authors, both in this country and abroad, upon this subject; how, by the simple experiment of blowing air into the lung, it was shown that sunken and airless lobules could often be re-inflated, their condition being one of collapse which even in life more vigour of inspiration might have overcome. This fact of lobular collapse once demonstrated, the mechanism by which it could be accomplished in the case in question was readily arrived at. With a new-born lung, hardly yet fully expanded, it was easy to believe that the presence of undue secretion in one or more of the larger air-tubes would suffice to shut off the lobules beyond it from access of air during the weak inspiratory effort. That done, the superior force of expiration, aided by the paroxysmal expiration of cough, would serve to expel such air as remained still imprisoned by the lifting of a supposed pellet of mucus, which thus, like a ball-valve, permitted egress only.

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[1] See Appendix E.

[2] Thus, for example, Dr. Gee, speaking of scarlatina in this relation, 'when bronchitis is fatal we always find pneumonia superadded.' 'Reynolds's System,' vol. ii. p. 344.

The site of these lobules and their wedge shape, the concurrent emphysema of the surrounding lung, the low vitality of the patients, and the invariable antecedence of bronchitis, all lent aid to this view. And thus, from the belief that these carnified spots of lung indicated an insidious extension of bronchial inflammation, there resulted (in this country from the labours chiefly of Gairdner, and Bayley, and West) a sharp revulsion. The condition came to be regarded as the natural concomitant of bronchitis in childhood, not an active and inflammatory condition, but a passive yielding and gradual obsolescence. Everything was explained by collapse, and lobular pneumonia for a while, in the estimation of pathologists, itself underwent that process.

Coexistence of inflammation and collapse.

Nevertheless it still remained to be seen whether this carnified lung, which the test of inflatability clearly proved to be collapsed, had undergone no other change anterior or subsequent to this. The fact of a lobule, or group of lobules, being so far pervious as to admit of forcible inflation was conclusive evidence against their air-sacs being filled to distension with a solid material; but it by no means excluded the notion that to some extent they were the seat of catarrhal or exudative products. That was still a question for minute anatomy. And minute anatomy thus appealed to has given answer that these two conditions, collapse and inflammation, are not opposed but nearly allied, that the one state tends to the other, that it is more common to find the two united than either alone.

Appearance of a lung so affected.

In regard to its anatomy, this secondary collapse of individual lobules in infantile bronchitis is more or less obvious according as the surrounding lung is more or less hyperæmic. Seen under favourable circumstances a lung so affected exhibits a number of purplish spots irregularly scattered, but commonly most numerous at its lower and depending part. These, varying in size from a hemp-seed to a walnut, with the number of lobules concerned, are firmer than the surrounding tissue. Such of them as immediately underlie the pleura give rise often to corresponding depressions in the lung's surface, thus indicating that the increased density is at the expense of diminished volume.

If now one of these dark patches be accurately cut out from the rest and examined by itself, it is found to resemble very much, in its weight and resistance to the fingers, fresh muscle. Hard pinching will extract from it only a little bloodstained juice which is quite airless. The surface of its section is smooth and uniform, except sometimes for minute bleeding points. In its toughness, its colour, and the want of real solidity, it differs altogether from the consolidated, fragile, and greyish lung of hepatisation. If that is to be named from its likeness to liver, this may be named from its likeness to spleen. By whatever name, it seems strange to us now that the close resemblance between this condition and that which the lung assumes when pressed into a corner by extreme hydrothorax, should have escaped its proper application, still more so that in a disease of commencing life the shape and size of these nodules should not have recalled the appearance of the unexpanded fœtal lung.

But observe next that these purple spots do not *all*

The collapsed to be distinguished from the filled lobules.

indicate collapse, only some of them. It often happens that re-inflation can be accomplished for some nodules but not for others, while in all alike, both those that do and those that do not admit of air being forced into them, a process of catarrhal proliferation is to be seen, a process of which the ultimate result must be to refill the collapsed air-sacs and expand these disused cells, not with air but with a new and solid material. In fact, so far from the presence of collapse disprоving the existence of these inflammatory products, it would seem in every case to render it probable, while the passage of the lobule, or collection of lobules, through that agency, from collapse to solidity is often almost within reach of demonstration.

For the description just given of the naked-eye appearance of this grossly speckled lung is not yet complete, or rather, as given, it applies only to a few cases. I have said that not all, but only some, of the nodules representing collapsed lobules are capable of re-inflation. It must be added that some of those that resist it fail also in other respects to exhibit the signs of collapsed lung. They are not tough, like spleen, they have become solid and friable, like true hepatisation, and, where position admits of the observation, in place of shrinking there is now swelling. The superficial nodules are not depressed below the general surface, but raised above it.

Varieties of lobular degenerative change.

It is, indeed, by the various modes of this catarrhal process, and the subsequent degradation of its products, that we get the many varieties of lobular degenerative change so minutely described by Dr. Wilson Fox.[1] Thus collapse and consolidation may be found side by side, or the central part of a carnified nodule will contain little solid masses embedded in its substance, and beginning to liquefy. Sometimes, together with nodules of carnification or simple collapse, a lung will contain distinct spots or points of a bright yellow colour, which when cut yield fluid or semifluid pus, leaving a minute ragged cavity, or bringing to view the dilated extremity of a small bronchus. To such appearances are superadded, in varying degrees, congestion and œdema of the intervening tissue, the bronchial mucous membrane is throughout red and vascular, and, on section of the lung, there is an abundant flow of frothy blood-stained serum.

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[1] P. 722 et seq., 'Reynolds's System,' vol. ii.

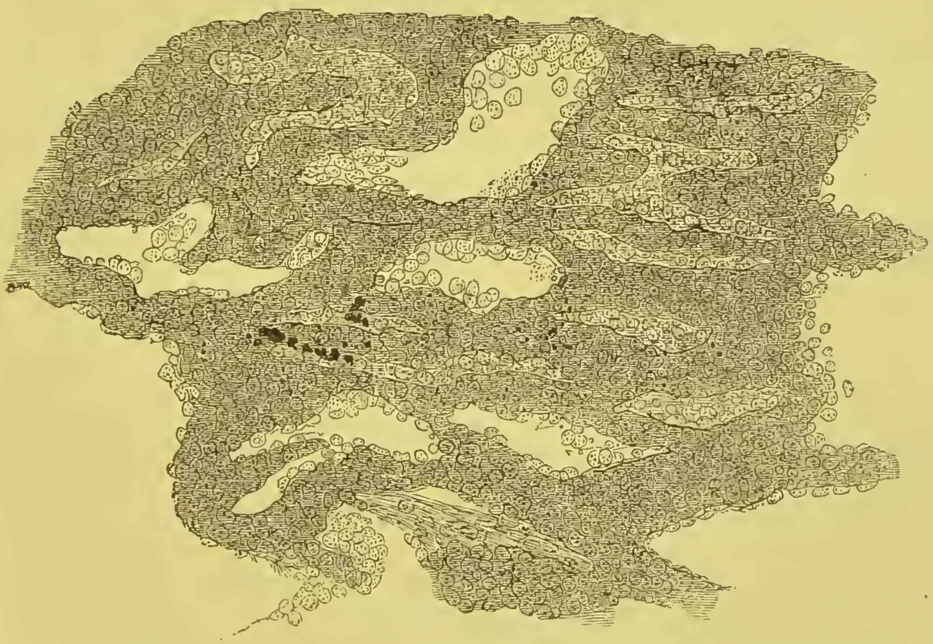

Fig. 3.—Collapsed portion of lung, showing proliferation from the alveolar walls, with commencing excavation. × 200.

Accidental resemblance to lobar pneumonia.

By this secondary participation of the lung as a whole the nodular appearance may become confused and indistinct, and the lobular arrangement is thus lost. It may even happen that the intervening outlying portions of the organ themselves ultimately become consolidated, and the lung at last (though representing a wholly different set of phenomena) comes to resemble that of lobar pneumonia. If to such a condition acute pleurisy succeeds, the general appearance of the lung may well be mistaken for pneumonia. In its inception it was distinctive enough, but at this late stage its individuality is lost. Yet even so late these variously altered spots are still traceable to mark the original affection and disclose its real nature.

Through all this variety of aspect certain features are discernible which, for our present purpose, claim particular attention.

Distinctive characters.

First, that this infantile lobular disease is necessarily associated (even when the fact of collapse is most complete and distinct) with cell proliferation, and that the existence of collapse of itself favours this further change.

Secondly, that this lobular catarrh, so far as we are able to observe it, is mainly deteriorative, and often destructive. These characters are exhibited in all stages of the process, from the early thickening and fibroid conversion of the alveolar walls to the final total destruction of entire lobules, which thus become converted into little cavities of fluid pus.

Relation to true pneumonia.

Thirdly, that in those nodules which eventually become solid, the contents of the separate alveoli, partly epithelial and partly corpuscular, are *in themselves* quite indistinguishable from the same elements as seen in pneumonia, so that, with all the difference existing between pneumonia and alveolar catarrh, the two at this stage have this one point in common.

Turning now for a moment to the clinical aspect of the subject, two questions naturally arise: first, as to the relation of this alveolar catarrh to the bronchitis which invariably accompanies it; secondly, as to the grounds for asserting that the affection is almost peculiar to infancy.

It may be asked, indeed, upon what authority do we

Symptoms of alveolar catarrh obscure or wanting.

give to alveolar catarrh, with or without consolidation, a history and a place amongst diseases when the marks of its progress during life are so indistinct and obscure. Obviously it must be hazardous to pronounce either upon the mode of origin or the measure of fatality of an affection which so often eludes us altogether. On what grounds, for instance, is it asserted that alveolar catarrh arises only out of a pre-existing bronchitis? Certainly not those of direct observation. It is an event constantly occurring, to be first apprised of this addition to the already recognised disease at the post-mortem table. In the instance of small patches of consolidation the auscultatory signs are habitually insufficient. Made watchful by experience, the increase of pyrexia and dyspnœa in an infant suffering with bronchitis may be taken as evidence of the implication of the alveoli. But there is absolutely no reason for saying that the advent of alveolar inflammation is always signalised in this way, or that a given degree of bodily heat will denote it. On the contrary, in many cases of hooping-cough and measles there is ground for supposing that *this alveolar implication is a very early event, and that it happens much more often than is commonly supposed.* In these two affections there is a peculiar liability to this particular pathological event. This is seen not only in the case of those infants that die at once, but still better in those that for a while seemingly recover and presently (the progressive alveolar destruction being finished) die of phthisis. It is seen as well—especially in measles—when softened spots like those that have been described—spots that stand for evidence of a prolonged destructive process completed—are found in the lungs of a child who has been but a few days acutely ill.

Relation to measles and hooping-cough.

In measles and hooping-cough, I say, *especially with a certain kind of children*, this lung affection is so common that it may almost be regarded in the light of a natural sequel. To these diseases it bears a relation similar in some respects to that between scarlatina and acute tubal nephritis. Thus, both the alveolar and the renal catarrh are associated with imperfect skin eruption, and both occur at the same period of life, while it is true of measles as of scarlatina that these two *sequelæ* respectively are common in some epidemics and rare in others. Yet the analogy fails in respect of the kind of subjects selected for attack. In the case of measles and hooping-cough, those infants suffer alveolar catarrh who possess the characteristics which are conveniently grouped together under the name of *scrofula*. In scarlatina, tubal nephritis follows no such rule.

Its connection with a particular diathesis, not with a particular period of life.

But secondly, and besides the liability which may arise from antecedent collapse, why is this pulmonary alveolar catarrh so common with young children? It is the custom to say that this frequency depends upon the fact that in infancy there is a special tendency to proliferate on the part of their epithelial elements. The remark may appear at first sight like a truism; an imposing way of saying, children suffer most because they are most prone to suffer. Yet in fact there is nothing in the circumstances to prove that this predisposition is not dependent rather on inherent constitution than on age or development. The affection, like acute tuberculosis, becomes associated with infancy as by an accident. It is a child's disease because it exhibits itself as soon as life begins, because the existence of this predisposition renders the continuance of life up to the adult period almost impossible. It is not that children are exceptionally liable to it as being children, but that those possessing this constitution in perfection seldom live to be men. The requisite predisposition existing there needs no more than the due provocation, say measles or hooping-cough, to make it operative. Add to this the fact that while at all ages alike the baneful effects of cold and wet are chiefly seen in exciting inflammation in the air-passages, little children are the least protected from such effects. It becomes unnecessary, then, to regard the pulmonary alveoli of these people as having special endowments differing from others.

The clinical and pathological differences between alveolar catarrh and true pneumonia.

A mere abstract of what has been said at tedious length will suffice to display the fundamental differences between this lobular affection and pneumonia. Clinically they would relate to its secondary origin from particular causes and in a particular class of subjects; to its stealthy development without constant or trustworthy signs of any kind; to its variable rate of progress and its liability to recur.[1] Pathologically they would relate to the origin of this alveolar catarrh in and from antecedent collapse; to the prolonged residence and the deportment of the catarrhal products, and the nature of those permanent changes which through this agency are ultimately wrought upon the texture of the lung.

Lobular collapse and its results in old age.

Before quitting this branch of the subject, it may be well to observe that alveolar catarrh affecting scattered lobules or patches of lung, in association with collapse, may occur at all periods of life.[2] The prolongation of bare existence with vitality lowered to its extremest point is a favouring condition. In this aspect the circumstances of the new-born weakly child and the aged and worn-out old man are not dissimilar. And accordingly collapse first, and subsequently the filling up of the alveoli from a localised catarrh may be observed at both ends of life.

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[1] With this secondary nodular consolidation of young children, the urinary chlorides do not disappear or even diminish. As to the urea discharge I have no knowledge. The determination of this point in these subjects, for obvious reasons, is exceedingly difficult.

[2] Dr. Peacock has lately written his opinion that 'the so-called "lobular pneumonia" of adults, essentially distinct from true pneumonia, consists really in collapse of the lung resulting from bronchitis.' 'St. Thomas's Hospital Reports,' vol. v., 1874, p. 1.

Purulent deposits scattered through the lungs in pyæmia.

It may be mentioned further that minute collections of pus scattered through both lungs and often described as 'abscesses,' may be met with in persons of all ages, so disposed as to resemble very nearly one of the results of destructive lobular inflammation as already described in the case of young children. The condition, which is but a form of pyæmia, is found in connection with uræmic poisoning and with phlebitis. Not seldom it is mistaken at the post-mortem table for softened tubercle, and in life is most often overlooked altogether. I make mention of it here only to ensure its distinction from the appearance it resembles.

CHAPTER X.

MORBID CONDITIONS OF THE LUNG DISTINGUISHABLE FROM PNEUMONIA—*continued.*

The indurated lung of cardiac disease—Clinical illustrations—Pulmonary obstruction from defective elimination—Hypostatic consolidation—Its anatomical and clinical characters—Wide relations of alveolar inflammation.

Indurated lung of cardiac obstruction.

THERE is yet another[1] morbid condition usurping the name of pneumonia, whose resemblance to that disease consists mainly in this, that after a series of changes in themselves distinctive enough, the lung becomes dense and tough, and in some instances eventually solid over a large area. The condition, which mostly affects both lungs, is best seen in the adult in cases of mitral cardiac obstruction, or, indeed, of any mechanical defect producing abiding pulmonary hyperæmia.

Its physical characters

In these circumstances there occurs a well-marked structural change strikingly opposed in its character to what takes place as a sequel to active inflammatory congestion. Grossly, this change is indicated by the tough, leathery consistence it confers. The finger cannot penetrate the lung, and, in extreme cases, the knife grates in cutting it. Though hardly solid it is not crepitant Its section shows a mottled surface of grey and brownish-red, with coarse white lines stretching across so as to map it out irregularly into groups of lobules. In this, its most characteristic stage, it feels like coarse muscle. The organ has lost its delicacy of texture. Essentially the same condition, as regards colour and fibrous intersecting bands, may be met with when in addition the lung is firmly solid; or, in the substance of a lobe which is tough only, a solid portion may be disclosed on section. Yet always the distinction remains to separate it easily from ordinary hepatisation in its greater resistance; it is not so solid but more coherent, breaking less but yielding more.

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[1] I do not include here other forms of indurated lung, commonly described *post mortem* as chronic pneumonia, which in their morbid anatomy differ from that described above. These are now usually classed with phthisis, and may be mentioned in that connection.

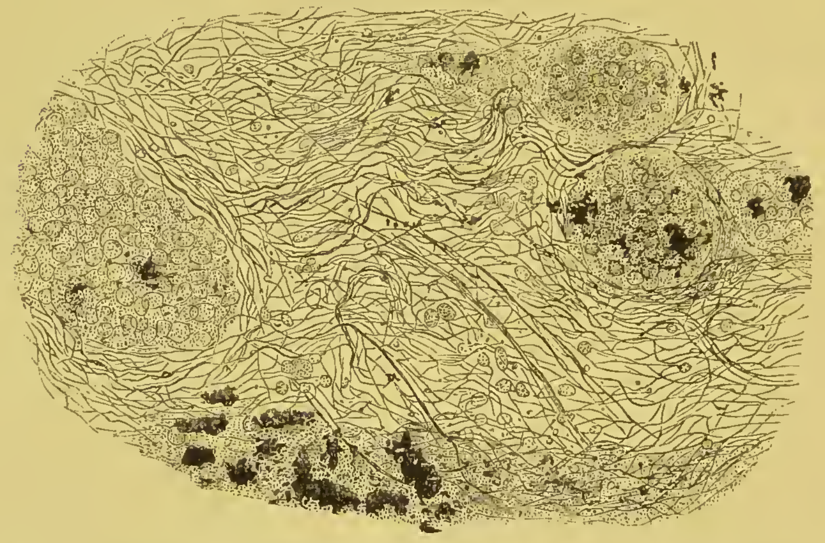

Fig. 4.—Minute appearance of tough resisting lung, from a case of mitral valvular disease, showing extensive fibroid development encircling the alveoli and separating them. The latter are seen with their contents partly granular, partly corpuscular, deeply pigmented. × 200.

and minute appearance.

In the microscopic examination of such lungs we encounter various appearances to one or other of which individual observers have confined their descriptions. These include increased and altered pigmentation, inter-alveolar amorphous deposits, fibroid metamorphosis producing thickening here and there of the alveolar walls, which in other places are no further changed than by the distension of their capillary vessels.[1] And here again, as we have learnt to expect, the alveolar spaces are loaded with catarrhal products.

The main feature, however, of the indurated lung consists in the increase of its fibroid elements, or rather, in the conversion of the normal elastic tissue into fibroid material. By the addition thus made to the normal texture of the lung air space is encroached upon. Inter-alveolar fibroid material is multiplied at the expense of the alveolar chambers which are thus in places almost obliterated. In an organ so far altered it needs only a slight increase or extension of the catarrhal process to render portions of it wholly airless and non-crepitant.

Further changes in splenisation and partial consolidation.

The agency by which this further change is accomplished is œdema. Through the intervention of a watery effusion, which itself results from impeded capillary circulation, air is displaced from the alveoli, and the lung, in those parts that œdema most affects, becomes carnified. At this period it resembles very much the compressed lung of hydrothorax, or the collapsed lobule of the child, as these appear in their later stage. And, as with them, this fluid exudation eventually gives place to variously-shaped elements mostly catarrhal, but with a variable intermixture of exudative products. Splenisation, however arrived at, is 'a favourable soil for the development of such changes.'[2] The lung is thus brought by a succession of steps, each arising naturally out of the other, to a condition of solidity more or less complete, which gets described as hepatisation and even attributed to acute inflammation.

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[1] See Dr. Wilson Fox's description under the title 'Brown induration of the lung,' 801, *loc. cit.*

[2] Rindfleisch uses this expression ('Path. Histology,' ii. 24), and I copy it. I would not of myself have ventured so far. The statement seems to require qualification, and to be somewhat roughly manufactured to cover the facts.

Anatomically distinct from pneumonia.

Yet no argument is needed to separate such phenomena from those of pneumonia. In the case we are considering it is the structure of the lung that is primarily affected; it has undergone a chronic irreparable change; the final filling up of the alveoli is a consecutive result which—though far from invariable—the altered conditions of circulation directly favour. In the pneumonia, on the contrary, normal alveoli are suddenly invaded by a plastic exudation, and the solidity which the lung acquires is due, not to itself, but to this added material.

Clinical characters.

The distinctive character of this condition is not less apparent from its clinical aspect. In Table III., Appendix D, will be found some short details concerning twenty-five patients, exhibiting upon inspection extensive consolidation of one or both lungs described at the time in every instance as pneumonia. The cases, it will be seen, are very various in duration, and symptoms, and time of life. Clinically speaking, most of them were 'chronic.' Sixteen of the patients were under thirty, the oldest was sixty-two. There is a distinct rheumatic history mentioned in ten. In the rest that point is not adverted to. With at least sixteen (or about two-thirds) both lungs participate in the change, generally as to the lower lobes, pretty equally and often in accurately corresponding situations. Pleurisy is exceptional. In those cases where the solidity is unilateral the fellow lung is described as 'infiltrated with serous fluid,' or congested, or containing patches of pulmonary apoplexy. Breaking down of the lung by purulent destruction is conspicuous by its absence.

Symmetry and absence of pleurisy and of suppuration.

Common cause.

Now in seventeen of these examples there is obstruction of a very definite kind, namely narrowing (in most instances in an extreme degree) of the mitral orifice. In the rest, though there is no direct obstruction in the sense of a material bar to the blood's passage, yet the circumstances warrant the assumption that a like effect is produced by the defective action of an embarrassed heart. In many of the patients a marked rheumatic history, together with youth, may fairly be presumed to have influenced the ultimate result; blood prone to deposit fibrin is placed in circumstances favourable to such an occurrence. Yet so manifest is the influence of a mechanical agency, that it seems sometimes as though an accident determined between exudation and rupture. The lower lobe of one lung will be solid, whilst its fellow is extremely congested, and contains in its midst a mass of extravasated blood.[1] Often, as might be supposed, there is general œdema of the limbs.

The precise condition of lung is not always minutely described in these records. Yet the coincidence of firmness and solidity with absence of purulent infiltration is sufficiently distinctive and remarkable. The terms used are 'solid,' 'firm and solid,' 'hepatised,' 'grey and quite solid,' and so forth, while the parts so changed are the same that suffer in ordinary hypostatic congestion. And what is specially noteworthy is the common absence of recent fibrin in the pleura. Sometimes we have fluid effusion, and once, viz. in Case 13, with more than half the lung in a state of 'red hepatisation,' the pleura is mentioned as healthy.

Some of these cases, in their leading incidents and by comparison one with the other, strikingly illustrate both the near dependence of the condition upon mechanical causes and also its essential chronicity or state of standstill; the structure of the lung exhibits great endurance and shows the same appearance after a week as after a month and a half.

Illustrations.

Take the following examples: A lad who for years has been subject to cough with occasional hæmoptysis is admitted with rheumatism and the 'physical symptoms' of pneumonia. He is bled, sinks rapidly, and dies in six days. The whole of the lower and part of the upper lobe of the left lung are hepatised, but the left pleura is healthy. The back of the right lung is in a similar state. The mitral orifice of the heart barely admits the tip of the forefinger. Decolourised clots occupy its chambers. A young woman has had cough and dyspnœa for six months, and later, dropsy. She is admitted with that and blood-spitting, and dies eventually with no acute symptoms after forty-six days. She, too, has the whole of the lower lobe of the right lung solid, and containing small patches of extravasation. The left lung is similarly though less affected. Here again the mitral orifice is narrowed so as only to admit the little finger. The heart is covered with old false membrane, and the ventricles uncontracted. A girl of 17, with obvious signs of heart-disease following rheumatism, emaciates, becomes œdematous and slowly dies. Both lungs are found to be hepatised, and the heart, much hypertrophied and adherent to its pericardium, is in an advanced stage of fatty degeneration.[1]

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[1] See, for instance, Case 12. See also, in abstract, a case of lung congestion, simulating pneumonia, consequent upon effusion of blood into the pericardium. Appendix C, Case 3, p. 256.

The obstruction may arise from the altered constitution of the blood,

While these cases thus exhibit a strong family likeness, it is not to be supposed that the effect upon the lung of these particular lesions is always as uniform as it appears in these selected instances. A number of causes will be in operation besides this obvious one of mechanical obstruction. Often the prime agent is not of a mechanical but, so to speak, of a vital kind. The freedom of circulation is impaired in the first instance, in accordance with a well-recognised law, by the deterioration of the blood itself. Thus the non-elimination of certain elements of secretion especially of urea, may be the first in the chain of causes that leads ultimately to pulmonary stasis. It is strictly accurate to say that such blood circulates ill, and conveys a morbid liability of that kind.

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[1] The Cases referred to are 13, 17, and 6 respectively, Class III., Appendix D.

Conceive then a weakened heart, with a firmly adherent pericardium, circulating this vitiated and, so to speak, unwilling blood through a mitral orifice narrowed to the point of the little finger. With such forces in operation it is easy to see where the strain would tell, and how, with each circuit of the blood, the evil would go on increasing. But when the end comes it is not easy—nay, it is manifestly impossible—to determine what part of the result was due to the bad blood and what to the faulty mechanism.[1] They must bear the blame together.

or mechanical imperfection may concur with defective elimination.

Lastly is to be mentioned, as wholly distinct from pneumonia, the hypostatic congestion to be observed in instances of prolonged dying. Here, too, consolidation is accomplished by the aid of œdema. With the expulsion of air, cell-forms, in themselves indistinguishable from the pneumonic, stealthily invade the alveoli. Consolidation (when that stage is reached) seeks the depending portions of both lungs, and has its situation sometimes curiously modified in correspondence with that which the patient assumed in his last hours. Not only will prolonged decubitus with extreme feebleness determine the fact of con-

Hypostatic consolidation.

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[1] The difficulties to be overcome in all such cases are indeed so manifest that one is apt to conceive of them too absolutely; to suppose that causes like these must be tending continuously to an ever-increasing pulmonary hyperæmia, the consequences of which were not merely probable but inevitable. Experience teaches otherwise. Individuals in the predicament we are supposing may go on for years without notable trouble. And more, they may become weakened by illness of an independent kind, yet this assailable point in their system will give no sign of surrender. It happens, for instance, often enough in actual experience that the heart which has repeatedly exhibited its weakness by sudden angina, perhaps during the repose and in the best health of its possessor, will hold out during the stress of a long illness or the strain of some unusual exertion. It is easier to understand all that these mechanical faults threaten, than to appreciate the nature or the worth of that vital compensation or reserve which is yet available to modify or supersede their operation.

solidation, but by placing the patient on this side or on that, or on the face instead of the back, we may settle beforehand the exact spot which shall become solid. 'The influence of decubitus,' says Grisolle, 'is so real that one can, so to speak, create secondary pneumonia at will in this or that part by varying the position.'[1]

Its anatomical character.

Pleurisy and hæmorrhage are rare with this condition of lung, while the gradual shading off of œdematous congested tissue into that which is carnified or solid as the parts are reached which lie most remote from the influence of the flagging circulation, is strongly suggestive of a single cause of disturbance reigning through the whole pulmonary system, and more or less operative in different places in obedience to the laws of physics.

Effusion into one or both pleuræ may concur with this form of consolidation, or may take its place. In the latter case the lung will be collapsed and leathery, and often contains solid spots here and there in its substance. The section of this solid portion is smooth instead of granular, and even in the most lengthened cases the passage into pus is exceptional.

Summary of thirty-nine such cases.

I have collected thirty-nine cases[2] from Hospital Records where long-standing illness terminated in lingering death[1] (phthisis being excluded), and exhibited in a marked degree this particular condition. The following is a short summary of these :—Twenty-two out of the thirty-nine had consolidation of both lungs ; ten of these were under thirty years of age, nine between that and fifty, and three are each fifty-four. None are aged, therefore. Of these same twenty-two, one only has recent pleurisy. Taking the numbers otherwise, and excluding from the whole two cases of erysipelas and nine of long-standing suppuration (since in these it might be urged that the appearances were due to erysipelas or pyæmia), there remain twenty-eight cases ; solidity affects both lungs in nineteen. The remaining nine have the one lung only solid, while the other is congested and œdematous. Again, of these twenty-eight selected cases six, at the most, give any intimation of purulent change. Three of these are instances of fever, two of cancer, and one is without history.

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[1] Pulmonary consolidation is spoken of by our own authorities as resulting from purely physical causes. Many years ago Dr. Williams wrote: 'It seems to me that the same mechanical congestion which sometimes leads to an effusion of blood in the tissue, constituting pulmonary apoplexy in other cases, if long continued enough, terminates in an effusion of lymph and an obliteration and consolidation of the pulmonary tissue.' * Stokes also, alluding to a passage in Andral † to a similar effect, says, 'We must agree with him in the opinion that the solidity of pneumonia arises, not from any deposition of lymph, but merely from an excessive congestion of blood.' ‡

[2] The cases are 46 in all, but 7 are rejected as ambiguous. I do not include this table with the rest in the Appendix, since the points it serves to illustrate may be as well stated summarily.

* Williams 'On the Lungs,' p. 145. † Spillan's 'Andral,' p. 378.
‡ Stokes 'On the Chest,' p. 312.

Consolidation dependent on age of patients.

Purulent change rare.

Great varieties in the nature and duration of the diseases ending with hypostatic consolidation.

It would be difficult to classify these cases clinically. They are no further alike than this, that they died, for the most part prematurely, worn out by the burden of some long-recognised disease, and that, so far as was observed, this tardy course towards the grave was not accelerated by the supervention of any acute or intercurrent affection.

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[1] It would be incorrect, I conceive, to conclude that this number represents the real frequency with which, in the circumstances stated, hypostatic congestion passes at last into consolidation. The subjects of lingering illness seek to die at home, or, wanting that, they are dismissed when treatment is abandoned to the workhouse. Still it must be admitted that consolidation is far less common with this mode of dying than the condition of œdematous congestion which according to the hypothesis immediately leads to it. It appears further, as shown above, that (having regard to the fact that the cases contemplated are most common in advanced life), this consecutive hypostatic consolidation selects rather the young and middle-aged than those advanced in years. Compare Appendix E for the statistics of children.

The list thus includes instances of malignant disease, of fever with exceptional lengthening out of life, of exhaustion after profuse hæmorrhage, of mechanical occlusion of the œsophagus entailing actual starvation.

Contrast for a moment acute pneumonia, as we all know it in its striking objective signs and busy eventful history, with this chronic process of deterioration, whose hidden course is regulated by the fluctuations of a wholly distinct disease. Contrast especially the part that pneumonia plays as a local affection, invading but not destroying and making the lung rather the field of its operations than the object of direct attack, with this gradual process of extinction which awaits the period of death-agony for its full development. However the two conditions may claim union on the ground of histological likeness or their common participation in an inflammatory process,[1] yet in origin, and course, and destiny, they are widely dissevered, and for all practical uses must be kept asunder.

We have here, then, a number of distinct processes of disease, each with its own history as to origin, and progress, and consequence, yet all knit together by virtue of common histological elements. Pneumonia, infantile lobular catarrh, scattered patches of consolidated lung surrounding gangrene or tubercle, no less than chronic induration or the solidity of passive congestion, all exhibit within the pulmonary alveoli not only those proliferative cell elements which we call epithelial, but escaped corpuscles also.

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[1] It may appear, perhaps, that in the endeavour here and elsewhere to restrict the term 'inflammation,' I am in fact only alluding to some of the circumstances under which it is allowed that certain products occur which pathologists are willing to recognise as inflammatory. I cannot think so. Inflammation cannot be thus partially recognised; it has a programme of its own, in which mere physical agency plays quite a subordinate part. 'A part,' says Prof. Simon, 'does not inflame because it receives more blood; it receives more blood because it is inflamed. The afflux is due to an influence primarily exerted by the part.'

And, indeed, examples of alveoli exhibiting catarrhal growth and corpuscular formation have a far wider range than this. The occurrence is, in fact, the commonest and the most invariable of all those that ensue where damage has been done to the lung, be the source of that damage what it may. In almost every case of injury, whether by disease or violence, the presence of alveolar catarrhal products may be predicted almost with certainty. In caseation, in tubercular infiltration, in grey granulation, in mechanical collapse, in the indurated lung of old valvular obstruction, there occurs, along with the special lesion which confers the name, a proliferation of supposed epithelial elements occupying the neighbouring alveoli. Intermixed very variously with these, and, to our present knowledge, anomalously, are those pyoid cells or corpuscles which we have been taught too exclusively to associate with pneumonia. The lung, or more truly the particular alveoli immediately implicated by this close neighbourhood, would seem always ready to respond in this way to any local irritation. Hence separate morbid conditions are anatomically distinguishable, *inter se*, not by this appearance, which is common to them all, but by other and coincident phenomena on the part of the several structures that compose the lung, as the alveolar walls, the supposed adenoid bodies, and the capillary vessels.

The wide relations of alveolar catarrh anatomically,

We may take for illustration caseation, or so-called tubercular infiltration, consisting not merely in a small-celled growth occupying the alveolar walls, but of epithelial products filling up the inculpated alveoli and indistinguishable from ordinary alveolar catarrh. Or take the grey granulation, true tubercle whose supposed origin is in a hyperplasia of pre-existing adenoid tissue; this too, though the retrospective eye contemplates it in imagination as once existing separately, is yet, as a matter of fact, hardly to be found except encompassed by alveoli similarly occupied.

illustrated by caseation,

grey granulation,

pulmonary gangrene, and morbid growths.

Speak of it as you will, as an inflammatory complication or what not, there it always is. It is the same with the pulmonary tissue immediately neighbouring a specific morbid growth, or a spot of gangrene, or an aneurismal tumour, or a dilated bronchus. Doubtless these same products are destined to play different parts in these several cases. The

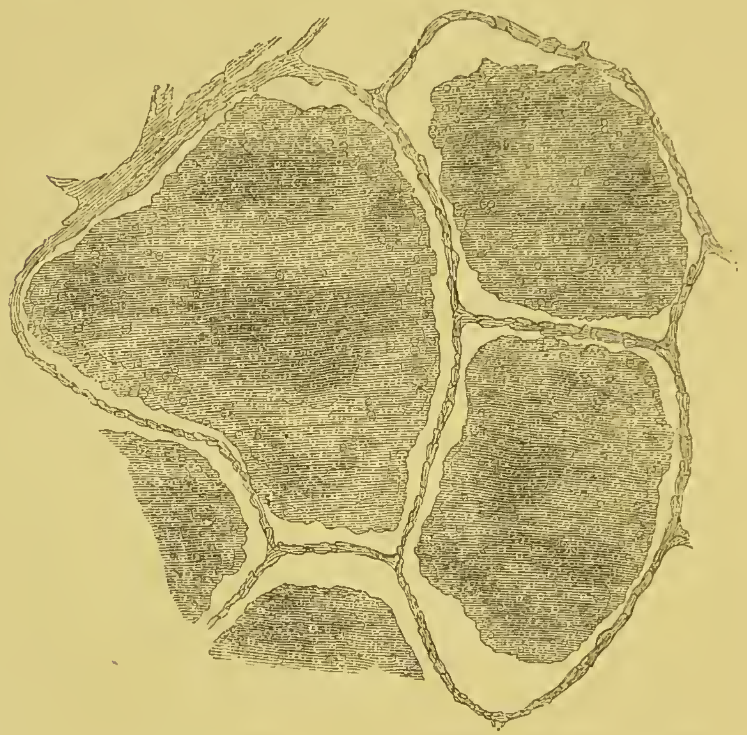

Fig. 5.—Alveoli in connection with chronic hepatisation and fibroid thickening at the apex of one lung in a case of diabetes of long duration, the patient sinking gradually *with no symptom whatever referable to the lungs.* The drawing exhibits alveoli filled with corpuscular material, the walls remaining unaltered.[1] × 200.

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[1] The woodcut is drawn from a specimen kindly lent to me by my friend Dr. Dickinson of St. George's Hospital, who describes the case whence it is taken in his treatise upon Diabetes, p. 157. It is drawn to the same scale as the other engravings for the sake of comparison, and the nature of the contents of the alveolus is thus obscured. But it shows sufficiently the point in question, and might do service as an illustration of ordinary pneumonia as described at p. 105.

same instruments variously used may work to divers ends. There they are, however. Whatever else is different this is the same.

Pneumonia not always distinguishable microscopically.

And observe, moreover, that these alveolar contents, thus widely related, are not necessarily wholly catarrhal. With the larger nucleated or epithelial cells there mingle, as has been said, smaller ovoid corpuscles like those which are the most characteristic of the formed elements of true pneumonia. And as in that disease, so here the latter occur in very varying proportions, sometimes exceeding the catarrhal contents, and sometimes even filling the alveoli almost to the exclusion of the others. Hence it may happen that a portion of lung viewed under the microscope fails to convey to the observer any certain impression as to the precise thing he is looking at. What he sees is but the common response to a local irritation of some kind or other. It may indicate equally either the most typical pneumonia or a condition which has no more than this in common with pneumonia.

CHAPTER XI.

THE INFLUENCE OF WEATHER AND CLIMATE IN PRODUCING PNEUMONIA.

Influence of temperature—of rainfall—of north and north-east winds—of humidity—Geographical distribution of pneumonia—General conclusions.

THE general influence of weather upon the so-called inflammatory diseases of the chest is matter of common observation. Low temperature, easterly winds, sudden changes of whatever kind, are believed to increase the whole group of chest affections alike. I propose to consider very shortly how far this popular view is the correct one; for it seems not unreasonable to expect that diseases so different in their history and progress as pneumonia and bronchitis should in their origin be influenced in different degrees by the same set of circumstances.

Uncertainty of conclusions.

Such an inquiry, to be complete, must review the weather conditions, both in this country and abroad, at several periods; at times when the diseases in question are unusually prevalent, and at times when they are unusually rare. Along with this, the geographical distribution of the two must be considered, and the records of past epidemics. Should any conclusion be arrived at by these means, it would still have to stand the test of our daily experience.

Even were the materials at hand for such an investigation, it would obviously be open to many sources of error. We have, for instance, to trust to reports of very unequal value. For our own country there exists no fuller information than that furnished by the Registrar-General's Reports, and for other countries there is hardly any information at all. And while we desire to deal only with the simple forms of the two diseases, there are no means, in such figures as we get, of separating these from the others. Further, it does not follow that the prevalence of a disease is always correctly measured by the number of deaths it occasions. There is, besides, the difficulty of estimating the period which should intervene between the weather, which is the assumed cause, and the death, which is the ultimate result. The mortality from pneumonia or from bronchitis suddenly rises; where precisely are we to look for the circumstances of weather to which that increase is due? In our ever-varying climate it must always be hard to determine this point, or even to say whether it is the weather of some particular period, or the mere fact of change of weather, which produces the result. These and numerous other sources of fallacy tend to disappear in a multiplication of instances. It is at least worth trying whether (regarding the Registrar's tables as true *comparatively*, and not at all for absolute numbers) it may be possible, by a series of observations upon the meteorological phenomena which attend periods of high and of low mortality from pneumonia, to arrive at some general conclusions as to the influence of weather; conclusions which must either stand or fall according to what we learn of the distribution of the disease throughout the globe, of the circumstances attending its epidemics, and of such histories of individual cases as our own experience may furnish.

General effect of cold seen most in bronchitis.

Accepting, then, thus far the information of the Registrar-General's Weekly Returns, it will be observed, in the first place, that under the influence of weather (and especially of cold) the whole number of acute chest affections are at least *similarly* affected; their death rates rise and fall together. The only question is whether, so far as this evidence goes, any particular condition of weather affects them unequally; so that, for instance, we may say that cold is especially prejudicial to one disease, easterly winds to another, and so on. In the case of pneumonia, the fact, which soon appears, that there is a near correspondence between its fluctuations and those of bronchitis adds, no doubt, to the difficulty of determining the precise conditions whose influence is unequal in the two cases. At the same time, if these two affections vary as to their death rate simultaneously, we may safely conclude that the causes on which these variations depend do the same; that they are to be looked for at one and the same time for both diseases. The objection, therefore, that to compare the death rate of bronchitis with that of pneumonia is to compare diseases differing in duration, which, if they end together must have started far apart, loses its force. In every week, no doubt, deaths occur under these two headings after very different periods of illness, and from causes quite remote from climate; yet on the whole, for purposes of comparison, we may assume from this strict harmony between the two that the fluctuations in the rates from week to week express truly the effects of the same period of weather in both cases.

Now it appears that the lowest mortality of both pneumonia and bronchitis falls in the latter part of summer, and the highest of both during the winter. The proportion of deaths, however, between the two diseases is not constant throughout the year. During the five or six temperate months, say from May to October, this proportion approaches nearest to 1, it being not unusual at midsummer for the mortality from pneumonia even to exceed that from bronchitis. But about the latter end of October or thereabouts, sooner or later according to temperature, the bronchitis rate rises in a greater degree than the pneumonia rate, and the maximum of difference for the two is obtained about January or February. It will be found, moreover, that while the minimum of deaths for both occurs at or about the same time, namely in August and September, the maximum of deaths is earlier in the case of pneumonia than in bronchitis. Thus, taking the average of ten years, 1857–66, the highest number for bronchitis occurs in the middle of January, while for pneumonia it is nearly two months earlier, that is at the end of November.[1] We have, therefore, these two diseases presented to us as of unequal range, that of bronchitis being the larger. In summer the causes provocative of both are at their weakest; as winter approaches these are more felt, and pneumonia is apt to suffer to its utmost earlier in the year than bronchitis.

Influence of epidemics.

Admitting, as all must do, that these changes are dependent in a great degree upon the changing seasons, it must be admitted, too, that the prevalence of other diseases, or of that general influence hostile to health which exists during epidemics, would tend from time to time to disturb the results arrived at by the mere comparison of one season with another. With communities, as with individuals, there are times, we know, when the system responds with exceptional readiness to influences which at other periods would be unfelt. In all the variations, therefore, of the death rate from bronchitis or pneumonia throughout the year, it is necessary to keep continually in view not only the same variations in other diseases, but also the general tone of the public health, and for every week to consider the Report on the whole, before attributing increased mortality to a direct weather influence. It does not appear, however, from the tables that a high rate of mortality from an epidemic always concurs with a high rate from bronchitis or pneumonia. In July 1865, for instance, when diarrhœa was more fatal than in the same month of many previous years, the mortality from the diseases in question was below the average. It appears, moreover, that during the cholera epidemic of 1849, and, less conspicuously, in that of 1854, there was a marked increase of mortality from pneumonia as compared with bronchitis. For the rest, we can say no more than that weeks of high mortality from typhus and 'infantile fever' are, as a rule, high for both these diseases alike, or so nearly alike that it would be unsafe to distinguish between them.

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[1] In Paris, according to Grisolle's observations, September yields the smallest, and April the largest number of cases of pneumonia. In Vienna the disease was most prevalent in the first five months of the year, especially in March, April, and May. See 'Reports of Vienna Hospital,' vol. i. 23, 1860, 61, 62, Schmidt's 'Jahrb.' Taking the numbers for Paris and London with a view to compare the rates from bronchitis and pneumonia respectively, we find often a striking difference in the proportions: thus—London, week ending March 12, 1870, bronchitis, 305; pneumonia, 100. Paris, week ending March 5, 1870, bronchitis, 100; pneumonia, 131.

To these considerations it must be added that in affections so allied as bronchitis and pneumonia—mistaken often the one for the other, or the name of either used indifferently to describe conditions for which there is no fit column in the returns—an increase in the one rate from whatever cause must always tend unduly to drag up the other. However unequal, therefore, the real influence of the several agencies now to be considered upon the two diseases respectively, the apparent inequality, judged of by the light of these tables, will always be less than the actual inequality.

Pneumonia somtimes unaffected by lowered temperature.

As regards the influence of temperature, it would seem that cold does not necessarily affect the pneumonia rate, but always and markedly the bronchitis rate. Take for instance the early winter of 1859: a low temperature towards the end of October and again in the middle of November is followed by a bronchitis rate greatly in excess of the average. Pneumonia on the other hand, though not unaffected by the change, is only slightly so, it remains to the end of the latter month much under its average; when with excessive cold and a north wind it rises considerably. Again, extreme cold in the middle of January 1867 is followed by a large increase in the rate of bronchitis, which for the week ending on the 26th rises to 162 over its average; while pneumonia actually decreases at the same time, and, to the end of the month, remains below its ten years' average; the direction of wind being north-west and south-west.

Bronchitis invariably increased by cold.

A glance at the tables will show how surely the rate of bronchitis is affected by cold. They show, no doubt, a similar tendency on the part of pneumonia; but, as has been shown, this is not invariable nor strongly marked. There seems, indeed, no assignable limit to the rise of bronchitis with lowered temperature. With the extreme cold of January 1864 its death rate (for the week ending January 16) is perhaps the highest on record; pneumonia, on the other hand, many times during the same year is higher than for this particular week.[1] It has been said already that pneumonia attains its maximum mortality in November. If cold were the chief agent in its production, it would, like bronchitis, be most frequent in January.

This conclusion as to the effect of cold receives support from what we know of the geographical distribution of pneumonia. It is a disease especially of temperate regions. At the poles and in the tropics it is very rare. It has been computed that the proportion of pneumonia to other diseases at Chamounix is as much as 1 in 5, while on the African coast it is as little as 1 in 627. It is very rare in the Presidency of Madras; equally so amongst those exposed to the extremest cold in expeditions to the Arctic regions.[1]

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[1] It may be urged with truth, that the general effect of extreme cold in carrying off old people is here to be taken into account, since these, although dying with some form or other of bronchorrhœa, are not in fact killed by bronchitis, although, for want of a better word, that cause of death is assigned. But even omitting these, or a large proportion of them, the above remarks are still true. It is the children, however, who contribute chiefly to swell the rate when cold is severe, and the great majority of these may fairly be claimed as the proper victims of the disease.

Rainfall

Does *rain* influence the rate of mortality from bronchitis or pneumonia? I have no sufficient data upon the subject, but such as I have yield a very uniform result. Take, for example, the year 1859. In the weeks following periods of abundant rain it happens always that the disproportion in the mortalities of the two diseases is conspicuous. Thus in the week ending July 2, 1·24 inches of rain fell; for the following week bronchitis is above and pneumonia below its average. In the week ending July 23 there were 2·15 inches of rain. For the subsequent week the mortality from bronchitis is the highest, and that from pneumonia is the lowest of the whole ten years of corresponding weeks quoted. There is, in fact—it so happens—only one week in this year where a great fall of rain is not followed by a similar altered proportion, and on this occasion nearly all the rain fell on one day, a steady downpour of nine hours. Take 1867. Here again it will be found that after weeks of unusual rain bronchitis increases out of proportion with pneumonia. Other years might be quoted to the same effect. There are, of course, numerous circumstances to be considered before asserting that wet weather has any direct influence of this kind; but I think no more than the truth is expressed by saying, that any considerable amount of wet has a tendency to heighten the bronchitis rate, but has no such tendency as regards the pneumonia rate; the very lowest numbers of this

affects bronchitis most.

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[1] 'British and Foreign Med.-Chir. Review,' xxii.

latter out of ten years being found to follow weeks of excessive rain (*Weekly Returns*, vol. xxviii. pp. 198 and 321).

Marshy districts and rainy season do not produce pneumonia.

As bearing upon this question, we find that in marshy localities, where intermittent fever is prevalent, pneumonia is little felt, although bronchial catarrh is often common in such places. We know, too, that during the rains of the monsoon in India pneumonia shows a remarkable decrease. According to an analysis by Dr. Morehead of 313 cases admitted into hospital, it appears that the three months of greatest rain—July, August, September—are the three also during which there is a remarkable decline in admissions from this cause. The numbers are the smallest of the whole year, and July, the very rainiest month, shows only nine such admissions, the next smallest number falling in August, and being just double of this. Dr. Morehead,[1] comparing his figures with those of M. Grisolle, observes that for these months there is a smaller decline for Bombay than for Paris ; he argues thence that a moist atmosphere and high winds tend to produce pneumonia. The inference is surely unjust. Dr. Morehead's tables render it probable that the range of pneumonia in Paris is greater than its range in Bombay. They show at the same time that the causes productive of the disease in Bombay are at their least during these monsoon months. If, then, rain is the great feature which distinguishes this season from others, we are forbidden to attribute to it any special influence for evil.

Wind.

In judging of the apparent influence of *wind*, it is not enough of course to regard only the quarter of the compass recorded by the register ; we must consider the force of the wind as well as its direction. At all seasons of the year there are many days when, from the air being comparatively at rest, we are unconscious of the fact that the wind, such as it is, is setting from the east or north-east; there is no wind properly so called. The proverbial belief of mankind which connects various ills to the body with the blowing of an east wind takes its origin from our own sensations when exposed to such blasts. It is not supposed—probably it is not true—that the same or similar effects follow when these sensations are not experienced. Calm weather or a small amount of horizontal movement of air is apt to correspond with low death rates from pneumonia, be the position of the weathercock what it will. Wind, on the other hand, it would seem, is favourable to the production of pneumonia, chiefly, if not altogether, when its direction is northerly or easterly. It is only an illustration of this remark to find, that during the calm weather of the spring of 1859, although the direction of the wind was registered as north-east, the mortality from pneumonia was unusually low. With the wind in that same quarter and a large amount of horizontal movement (as in March of 1867 and August of 1866) the mortality greatly exceeds the average. If we say, then, that high winds from the north and north-east, combined with a low temperature, yet not necessarily very low, favour the development of pneumonia, and that bronchitis, while similarly affected by the same causes, suffers in a less degree, I think the statement will be borne out by the returns. It may be added, that the effects of a very low temperature and of much rain are seen in raising the bronchitis rate rather than the pneumonia rate.

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[1] Dr. Morehead on the 'Diseases of India,' vol. ii. p. 308 et seq.

The influence of north and north-east winds.

Illustrations.

It may be said that observations on several agencies taken separately are open to objections. Let us take them in combination. We may choose, for instance, a year of very high mortality so far as these diseases are concerned, and compare it with one of very low mortality, and so, by placing the weather phenomena of the two years side by side, discover the conditions on whose presence or absence the variations in the death rate apparently depend. Now it happens that the summer of 1859 was remarkable for a very low mortality from pneumonia, and the summer of 1866 for a very high mortality from the same cause. In the former year the bronchitis rate is not affected; in the latter it is high, but not raised in an equal proportion with pneumonia. As regards the state of the general health, the total of deaths for both years exceeds the average, owing to a large mortality from diarrhœa. In 1866 there was a short outbreak of cholera, which, commencing in July, was at its height at the opening of August, when 1,053 deaths are registered for one week. From that time it declines considerably, but still numbers between 100 and 200 died weekly until late in the autumn. At the worst of the epidemic we have pneumonia showing a higher mortality than bronchitis, both being above the average. A similar phenomenon is observable in other cholera years, and especially in 1849.[1] On the other hand, in the year 1859, with a severe epidemic of diarrhœa, but with very little cholera, there is an almost unprecedented fall in pneumonia, in which bronchitis does not share.[2]

1859 compared with 1866.

What are the meteorological circumstances concurring with the low and the high rate specially affecting pneumonia in these two years respectively? Both summers are exceptionally hot. With the low rate of 1859 the wind is chiefly southerly, less variable, and for a fewer number of days easterly and north-easterly than with the high rate of 1866. The horizontal movement of air throughout the months referred to, July and August, is much less for the healthy year. Furthermore, with the year of high mortality, the wind veers round to the south-west in the week ending September 8, and the pneumonia rate becomes less for the week following. In fact, the great disparity between the rates of the two years disappears for a while at this point, and so long as, for both years, south-west winds are blowing. In October, however, with a return to the conditions of the summer, variable east winds against less variable south winds, there is again a wide difference. The healthier autumn is also much the colder; south-west winds and a small horizontal movement still coincide with the low rate.

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[1] See, for instance, weeks ending August 4 and August 11 of 1849.

[2] See especially weeks ending July 30 and August 13, 1859.

Comparison of corresponding weeks of separate years.

Again, place the week ending November 12, 1859, beside the week ending on the same day of 1864. I choose these two only because they exhibit the largest difference I can find; for 1859 the deaths from pneumonia are 60, for 1864 they are 165. Bronchitis, again, does not sympathise in the case of the low rate, but it does in the case of the high. There is no severe epidemic in either year, but typhus is twice as fatal in 1864 as in 1859. Taking the weather of the latter part of October in each of these years, it appears once more that the healthier year for pneumonia is by far the colder—the temperature indeed of the week ending October 29 is only 38·6°; it is unprecedentedly low—far lower at least than any other year shown. The effect of this low temperature is not unmarked; pneumonia rises considerably, yet for one week only, and then not so high as to reach its average; bronchitis, on the contrary, rises from much below to much above its average, and continues to rise during subsequent weeks. From the middle of October to November 5, 1859, the period of the low rate, the wind is chiefly south-west. That period, therefore, contrasts remarkably with the high rate of the corresponding time in 1864, when the direction of the wind is north and north-east. There is more rain with the low pneumonia rate than with the high.

Once more, we find included in one and the same year —the year 1868—a remarkable contrast in comparing similar seasons, February showing a very low rate from pneumonia, and December a very high rate. Thus the deaths from pneumonia for the week ending February 15 are 58, and from then till the end of March the weekly numbers are just over 70. In December, on the contrary, the rates for three successive weeks are together 360, as against 207 for three weeks of February. Now the temperature during the last weeks of January and of November (the periods respectively preceding the low and the high rates) is about the same, but the beginning of February is colder than the beginning of December; still, therefore, we have the greater cold with the healthier time. The direction of the wind from January 26 to February 15, that is with the low rate, is mostly south-west and west-south-west. It is the same during March; and during March there is again a low mortality in pneumonia.

Fluctuations in the death rate followed through a year.

If the weather for this same year be followed throughout a similar rule seems to prevail. Thus for the first half of April the wind is easterly; by the 18th of that month the mortality has risen to 98. As regards the high rate which distinguishes the latter part of the same year, we have the wind variable from November 22 till the end of the month, then east and south-east till December 3, when, after a few days of south-westerly winds, it again becomes variable, but chiefly south-east till the 15th. At this date it sets south-west, and continues in that quarter with hardly a break till the end of the year. And now the supposition that this heightened mortality of early December is somehow connected with the direction of the wind is rendered probable by the fact, that with the change to south-west we have for the last two weeks of December—colder weeks and very rainy—a mortality of 170 against 243 for the first two weeks.

It is satisfactory to find in corroboration of these state-

Similar statistics for Ireland.

ments that the Returns of the Registrar-General for Ireland are very similar. Drs. Grimshaw and Moore, who with much pains have investigated this subject,[1] find that for the four quarters of the year the mortality from bronchitis is markedly influenced by temperature; it was twice as great in the first as in the second quarter, and more than four times as great in the first as in the third quarter. The mortality from pneumonia, however, was only one-fifth greater in the first than in the third quarter. The extreme winter fatality of bronchitis and its low summer fatality are equally wanting in the case of pneumonia.

The further statistics of these authors in reference to Dublin, as regards the influence of rain-fall and of dry north-easterly winds, are still in strict accord with mine.

Dr. Peacock's evidence.

Hospital statistics, though in themselves much more reliable than these, exhibit numbers too small to be safely dealt with. Speaking generally, the prevalence of pneumonia during the summer is no uncommon experience in our London hospitals. Of one of them—St. Thomas's—Dr. Peacock says:[2] 'In the last fourteen years the largest number of admissions was during spring and summer. Taking a more limited period, 10 cases were admitted in the winter quarter, against 20 in the spring and 22 in the summer quarter: facts pointing,' as Dr. Peacock believes, 'to the production of the disease rather by sudden alternations of temperature and chills when the functions of the skin are active, than to severe, though more continuous cold.'

These details are tedious, and must fail to be wholly convincing. There is a difficulty in rendering them even intelligible without appending the tables to which they refer. Such as they are, the conclusions to which they point seem to be in accordance with the little that is to be learnt regarding the distribution of pneumonia throughout the globe. We know that neither extremes of temperature, nor swamp, nor moisture, nor the climates which in the world are most unfriendly to human life, have any marked influence over the disease in question. It is in the regions that are called temperate and in their exposed places that pneumonia is especially prevalent. Where north and north-east winds prevail, where the configuration of the country is favourable to currents of cold air, in such places as Madrid, Genoa, Florence, Naples, Gibraltar—different in other respects, but alike in this—in the high plateaux of countries whose lowlands are strangers to the disease, as in Africa and Mexico, pneumonia finds its chief victims. Shall we be very far wrong, then, in concluding that the brisk movement of air at a moderately low temperature (which perhaps might be proximately determined in degrees) is amongst the most obvious exciting causes of simple pneumonia—cold, dry, penetrating winds from the north and north-east for pneumonia; a greater degree of cold, wet weather, variable winds, for bronchitis?[1]

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[1] 'Dublin Journal of Medical Sciences,' March 1875.

[2] 'St. Thomas's Hospital Reports,' vol. v. p. 5. At the time of writing (June 1875) pneumonia has been more abundant in the Westminster Hospital than at any other time of the year.

Geographical distribution.

The frequency of pneumonia at great elevations may depend upon conditions which I do not now discuss. We are told that pneumonia and pleurisy take the first rank amongst the diseases of the most elevated towns and villages of Europe. From numerous observations M. Lombard is led to believe, that the prevalence of inflammatory maladies of the chest is in direct proportion to the elevation of the place above the sea-level. It is stated further, that the spring is their time of greatest frequency; the sickness increases as the snow melts. Moreover, epidemics of pneumonia, such as have been often observed in the mountains of Switzerland, belong more to the high valleys than the low, whilst the reverse is the case with regard to epidemic catarrh.[1]

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[1] It is remarkable that at Gibraltar, where pneumonia prevails during the greater part of the year, the west winds of November and January concur with a greater frequency of the disease than do the east winds of other months. But then the east winds are described as bringing along with them much humidity and fog, while the west winds are cold and dry. Grisolle, 'De la Pneumonie,' p. 124.

Circumstances of epidemics.

But the evidence from geographical position, and the evidence from our own statistical tables—supposing both to be as complete as possible—must still be supplemented by accurate observations upon particular epidemics and individual cases. For the first I may refer again to the circumstances of the epidemic occurring in the 22nd Regiment at New Brunswick.[2] It was remarkable in this instance, first, that in the coldest month of the three over which the epidemic spread, the admissions into hospital from pneumonia were much fewer than for the following and warmer month; and, secondly, that that portion only of the regiment suffered which was quartered in a new building abounding in draughts. Among the women and children, who were better housed, only two cases of pneumonia occurred.

Evidence of individual cases.

For the testimony of individual cases, perhaps the most convincing of all, there are those histories of simple pneumonia where we have before us both the proof which post-mortem inspection affords that the disease was primary and uncombined, and along with that strong reason to believe that it was immediately excited by some weather cause. I have already quoted cases of this kind, and may refer to others in the Appendix, where the sudden fall from health has been strikingly connected with some agency of this kind.

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[1] Lombard's 'Climats de Montagnes,' p. 70 et seq.

[2] See the admirable paper by Assist.-Surg. Welsh, 'Army Medical Reports for 1867,' p. 329.

Provoking causes alone discoverable.

Observations such as these are likely ever to fall far short of demonstration, they refer to a small part only of a very large subject. The etiology of pneumonia must comprehend all antecedent states of the body which render it assailable from without. And, however it may seem to us that the disease is brought about by such circumstances of weather as those I have been trying to investigate, we know that it is not wholly so ; that its root lies deeper than these, that they do but call it into life. Often, indeed, it is utterly beyond any reasonable conjecture to assign to simple pneumonia *any* external exciting cause. A number of antecedents have an equal claim in this respect. And accordingly, in common with every other diseased state, many things have been charged with provoking it upon evidence even more imperfect than that which has been adduced in the present chapter.

CHAPTER XII.

THE PATHOLOGY OF PNEUMONIA.

Pneumonia an inflammation—Its distinctive characters—Pyrexia in relation to the lung changes—Exciting and predisposing causes—Likeness to quinsy—Relation to rheumatism—Association with kidney disease—Origin in defective elimination in certain subjects—The place of pneumonia among diseases.

In the foregoing chapters I have been led to consider both the clinical phenomena of pneumonia and the several morbid changes with which these concur. It will be possible now to regard the affection as a whole, with a view to determine its nature upon this joint evidence.

Pneumonia an inflammation.

Whatever opinion we may be disposed to take of the proper scope and limits of true pneumonia, it will at least be admitted that in the typical form of the disease the phenomena of inflammation are conspicuously exhibited. Both the clinical and the anatomical requirements are satisfied. There is pyrexia, there is exudation with the migration of leucocytes, and there is excess of tissue change by proliferation. Not only so, but the fibrinous exudations which attend pneumonia, the pleurisy which is constant, and the pericarditis which is not rare, bear similar testimony, which is still further corroborated by the 'sthenic' constitution of the patients themselves and the character of the allied diseases. Not only is pneumonia an inflammation, it is the pattern and model of inflammations, and, in the history of medicine, has often been referred to in that character.

Its distinctive characters.

It is to be noted, at the same time, that pneumonia is not an inflammation merely, or a process that can be induced by artificial means. Neither by wounds nor irritant inhalations, nor in any other way, can the disease be set up, or anything at all resembling it. It must come. Nor have we here, as in the case of traumatic inflammations, a local change first, and next, as in response to this, a set of phenomena which are to be regarded as its proper consequence. On the contrary, the pyrexia will sometimes, as we have seen, precede, beyond all question and by several days, the earliest physical signs of change within itself.[1]

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[1] This antecedence of pyrexia (already adverted to at p. 33) is regarded by some as apparent and not real. The local affection, say they, is so centrally situated that its sounds do not reach the ear, or reaching it they fail to be recognised. The real first stage of pneumonia, according to these, is not crepitation, but harsh respiration only. If, therefore, this earliest sign be overlooked the commencement of physical change within the lung will be post-dated. Allowing the utmost validity to such arguments, they are insufficient for their present purpose. As for the cases in which the local affection commences in a deep-seated part of the lung, we can afford to exclude them altogether, the antecedence of pyrexia is not limited to these, nor indeed is it observed of them more than of the others. But admitting the fact that harsh respiration precedes by a little the sound of crepitation, the period of duration of such respiration would still be far too short to occupy the required interval. We may allow the harsh respiration; no one would venture to assert of it that it may last alone for two or three days. There are, indeed, some reasons for believing that the time which may intervene between the general and the local symptoms of pneumonia is commonly understated, owing to the position occupied by bronchitis in relation to the disease. It is well known that in affections which do not immediately concern the lungs, the occurrence of bronchial catarrh along with the initial pyrexia is not unfrequent. It happens so in typhoid, and still more in typhus, where it often precedes those special signs which ultimately determine the real character of the disorder. But with pneumonia it is different. Bronchial catarrh is here taken to indicate the commencement of the local mischief, and if presently the proper signs of pneumonia are recognised it is loosely assumed that these have been present through-

Relation of pyrexia to the lung changes.

Nor, in their further progress, is there any precise correspondence between the pyrexia and the local affection. The one is sudden in its origin, regular, or at least conforming more or less to a rule in its course, and abrupt in its departure; the other advances by degrees, unequally in different parts, and, having attained a certain point, retires, as it came, by gradual resolution. In no certain or constant relation to the first access of pyrĕxia, an exudation takes place which, becoming more and more fibrinous, soon solidifies and becomes moulded into its place. Often the area of lung at first affected widens as the disease goes on, and sometimes from a small patch of lung the process spreads so as to involve nearly the whole of it. Whether this is so or not, it is seldom that the part affected, be it large or small, is affected throughout simultaneously. While one portion is œdematous, another is sealed up with solid material, and another beginning to discharge its alveoli of their now liquefied contents, or perhaps to soften by a destructive ulceration. True, the whole portion concerned is implicated similarly, but the different parts exhibit different stages of one process. There is thus no moment when the local disease passes as a whole from hyperæmia to consolidation, or as a whole from red hepatisation to grey infiltration.

The same may be said for the process of restitution. This latter, indeed, is apt to be more tardy than the other, and even to remain incomplete when the health of the patient is pretty well restored. The lung clears up by degrees, and bit by bit.

Now, from this coincidence of several stages or degrees of solidity in the same lung, we should be led to expect, on

out, and that the bronchial catarrh was in some sort their first exponent, as though bronchial catarrh and pneumonia were not perfectly distinct and dissimilar.

the hypothesis of a local inflammation, not a definite course of pyrexia, but a series of elevations and depressions succeeding each other quite irregularly, according as fresh portions of lung became implicated, or as the local changes proceeded faster at one spot and slower at another. And not only would the pattern of any temperature-chart indicative of the natural course of lung inflammation be continually disfigured by this concurrence of its several stages, but sometimes its proper phenomena ought to repeat themselves. In double pneumonia, for instance, so soon as the second lung began to suffer, as its fellow had suffered before it, we might expect, but for the teaching of experience, a second rigor, together with a recurrence of the early temperatures. We see no such thing. The local inflammation, in its gradual extension and composite characters, offers no sort of parallelism to the fever which for a while accompanies it.

It is a further illustration of the distinctive and, so to speak, independent character of this pneumonic pyrexia, that its duration is usually limited, that it does not subside by degrees but stops of a sudden. In an ordinary case of pneumonia it is while the local affection is gradually undergoing a succession of orderly changes, often it is while the process of solidification, so far as we can judge by sounds, is yet progressing, that the fever terminates and the acute features of the disease all at once disappear.

Pneumonia not a local inflammation merely.

Such considerations as these are opposed to the view that pneumonia is nothing more than a local inflammation; they lend support to the belief that it is to be regarded rather as a specific disease having its chief seat in the lung. Other points might be taken in corroboration. We are not ignorant of the manner in which the system suffers in direct response to pulmonary lesions which in their physical results are similar to pneumonia. In these circumstances, as we have seen, the phenomena of the disease in question are not even imitated. It needs only to recall the look of pneumonia to concede as much as this.

Is it a specific disease?

What is it, then? Not a specific disease, say some, for it owns no specific cause. 'The causes of pneumonia,' says Dr. Wilson Fox,[1] 'are manifold, and the disease may originate under such diverse conditions that the theory of a specific cause can hardly be maintained.' In the wide sense in which the term is applied by this author these conditions are indeed diverse, as I have endeavoured to point out. But does not this very diversity furnish an additional argument in favour of a stricter limitation of the word than Dr. Fox will admit? Substitute only for pneumonia, in the sentence just quoted, the word hepatisation, or alveolar catarrh, or lobular consolidation, and the truth of the statement becomes apparent. These conditions—all of them in the histological sense inflammatory—are various in their mode of origin as in all other respects. But it is not so with pneumonia. In this relation, as in others, it is able to maintain its claim to separate and distinct recognition.

Its exciting and predisposing causes.

We know little enough of the mode of origin of any disease, no more, indeed, than some of the circumstances which sometimes favour its development. As much as this is known of pneumonia, but the knowledge is obscured and distorted by the intrusion of irrelevant examples. That it occurs in the greatest perfection with the young and robust, that certain constitutional states exhibit it most, that particular diseases are incompatible with it, that climates like our own, and certain states of weather, obviously favour it—these are facts admitted for acute sthenic pneumonia, but little insisted upon on the ground that they are inapplicable to the disease in its entirety. Thus, as the anatomical view of pneumonia is extended, and the affection is regarded exclusively from that side, its several boundaries disappear one by one, until at length all attempts at definition become impossible.

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[1] Dr. Fox in Reynolds's 'System,' ii. 675.

Its likeness to the specific fevers

But further, regarding pneumonia for the moment, as a provisional hypothesis, in the light of a specific disease, by reason of its mode of origin and class of subjects and particular phenomena, let us consider its likeness or unlikeness to other affections of the same class. Compared with the fevers, its points of difference are at least as conspicuous as its points of resemblance. Although a general disease, with a history accomplished within a given number of days, it is not contagious; it is not, in a strict sense, epidemic; it has no fixed 'prodromata,' or days of incubation.[1]

But although pneumonia, in its habit of announcing it-

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[1] We shall only obscure the truth in this matter by accepting the aid of ancient accounts of pneumonia. These no doubt describe true epidemics, but from the post-mortem evidence, scanty as it is, it would seem that they do not describe true pneumonia but rather congestive bronchitis. As for the pneumonia of our day, although its frequency varies from year to year, and circumstances of weather may happen to be so combined as to render it prevalent or the reverse, yet it is not epidemic in the same sense as scarlatina or small-pox or influenza-catarrh are epidemic. A true epidemic confers a liability to sicken after a particular fashion which is independent, or at least in excess, of the liability attributable to recognised causes, such as temperature, dwelling, and climate. These may still influence it, but the epidemic influence is paramount and apparent under all combinations of these. Thus the great historical epidemics desolated whole countries, and for the time almost rendered animal life impossible. Although likened to known diseases they always had besides something special about them, they were now more and now less malignant from wholly unknown causes. A combination of circumstances may render any disease excessively common or excessively rare, but it is only certain diseases that are observed from time to time to appear in an exceptional epidemic shape; to arise, to culminate, and then slowly to retire, affecting populations indiscriminately when the circumstances which are known to favour their spread in ordinary times are not present. Pneumonia is not one of these.

and to disease excited by a specific poison.

self suddenly in all its completeness, is quite out of the pattern of continued fever, it may nevertheless own a specific cause, and depend for its development on some anterior change which is distinct and invariable. What resemblance can be closer for two diseases quite separate as to their actual pathology, than the sudden stitch or rigor which, in the case of pneumonia, rudely interrupts a condition of apparent health, and the acute agony in the great toe which, with no sort of warning, at once plunges its victim into all the troubles of acute gout. True, gout does not always happen so. With a deeper insight than we possess in the present case, we are enabled to detect it, apart from its paroxysms, under various so-called disguises, and are thus indifferent as to the particular form in which it presents itself. We may know as much some day of the real relations of pneumonia, and be enabled to enlarge its realm accordingly.

Its clinical phenomena similar to those of quinsy.

The disease that pneumonia most nearly resembles, whether by accident or real kindred, is quinsy. In the character and duration of pyrexia, the rapid recovery or 'defervescence,' the severity of the general as compared with the local symptoms, and the age of the patients, the two affections are strikingly similar. Aching limbs, prostration, chilliness, and often distinct rigor, accompany or sometimes precede a quinsy where the local inflammation is quite insignificant. And not only is the pyrexia well marked (the thermometer always, according to Dr. Squarey,[1] rising above 102°), but the course of it, in respect of an early rise to its extreme height and rapid 'critical' decline about the fifth or sixth day, resembles more nearly than any other disease the temperature tracing of pneumonic fever.[2] The diminution of chlorides in the urine and increased urea discharge are mentioned as further points of likeness.

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[1] Reynolds, *loc. cit.* iii. 35.

[2] Wunderlich, 386.

And observe, moreover, just as pneumonia differs from the various forms of pulmonary consolidation, so does true quinsy differ from other forms of tonsillitis. It is a familiar experience to observe one or both tonsils undergo a gradual increase in size, and at length even suppurate, while no active disturbance accompanies the change, nor indeed any symptoms whatever except those that arise immediately from mechanical causes.

Relation to herpes.

This parallelism between quinsy and pneumonia seems further established in the relation of both to herpes. For as herpes about the lips and nostrils is a not infrequent accompaniment of pneumonia, and sometimes serves even to identify it, so does a precisely similar eruption of vesicles affect sometimes the pharynx and tonsils. The course of this herpetic tonsillitis is similar to that of quinsy, and occasionally pneumonia concurs with it.[1]

But it is less by means of analogies like these, which may easily be overstrained, than in the investigation of the actual circumstances of the disease itself, that we must hope to discover its real place. Here, as elsewhere, we shall be ultimately relegated to the bare region of hypothesis. Pneumonia is the product of a '*materies morbi*, and its special seat is the *locus minoris resistentiæ*.' Yet before seeking this final refuge in mediæval conceptions and a dead language, there are certain definite observations regarding the composition and the changes of the blood in pneumonia which may help to determine its nature.

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[1] It may perhaps strike the reader that herpes itself in its form of herpes zoster or shingles has a certain analogy with the disease we are discussing, in its unilateral site, its preference for the right side, its limited duration, course of temperature, and spontaneous departure. It appears also under two forms; sometimes it is preceded and accompanied by intense pain and high fever, at others it is quite free from these, and may even be overlooked.

Changes in the blood in pneumonia.

It appears from the investigations of Andral, which later observers have confirmed, that, as the pneumonic process goes on, so the amount of fibrin obtainable from the blood increases. This excess, which is not always noticeable at the commencement of the disease, becomes more and more pronounced as it proceeds, and is at its height on the eighth or ninth day. The phenomenon is not dependent on bloodletting, for it was observed by Zimmermann[1] in cases bled but once each, viz. on the third, fourth, and eighth days respectively. The later the bleeding, the greater was the amount of fibrin.

Thus (contrary to what might have been expected), it is after the occurrence of fibrinous exudation, and when fibrin has been largely yielded up to the lung, that hyperinosis is the most extreme. The exudation is from blood overcharged with fibrin-producing elements, but with its occurrence that quality of the fluid does not disappear, but rather increases. It would even seem that at the outset of the local inflammation and the height of pyrexia the blood was in this respect normal, and that the most marked hyperinosis was simultaneous with the completion of exudation, and not perceptibly diminished by that discharge.[2]

Pyrexia in relation to hyperinosis exudation.

Hence it would appear that the pyrexia and these local events of inflammation are in no direct relation to each other. The former is often most marked before the latter have appeared, and completed while these are yet in progress. We know, further, that in other diseases extreme pyrexia may occur where the fibrin to be yielded by the blood is not in excess but in defect. It is so in typhoid, in small-pox, in glanders.[1]

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[1] Andral, Ess. Hoem. Path., p. 87. Zimmermann, Prater Vierteljahresch., vol. xxxv., quoted by Fox, *loc. cit.*, p. 675.

[2] Thus Zimmermann found the fibrin increased to 9·10 per thousand on the eighth day, while in eight cases of commencing pneumonia the blood yielded either a normal or less than normal amount of fibrin.

While hyperinosis is always accompanied by high temperature, high temperature is not always due to hyperinosis; it may occur with the contrary condition, and probably is never absent when the constitution of the blood is anyhow suddenly altered.

Phenomena due to the fact of hepatisation.

Now it must not escape notice that the pneumonic process, considered as to its local part, is of the nature of a hæmorrhage, that it implies the transference from the circulation of a considerable portion of its material. The firm hepatised lung, outweighing its fellow by some three pounds,[2] represents a condition similar in some respects to that obtained by the actual abstraction of blood with the lancet. Now, just as it is found that with successive bleedings the blood becomes thereby not less but more 'fibrinous,' so it may be here. The hyperinosis, associated as it is rather with hepatisation than with the initial fever, may in fact be directly favoured by that event. Excess of fibrin, though it is observed in pneumonia most, is seen also in exudative inflammation everywhere; in simple pleurisy, in pericarditis, in fibrinous exudation from the kidney.[3] It is as though the fibrinous discharge which is common to all these affections were inadequate. The elements in excess accumulate in the blood notwithstanding, and the morbid deposit which is the expression of that accumulation relieves it but partially.

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[1] Dr. Woodman, in his translation of Wunderlich (*loc. cit.* p. 142) quotes temperatures in typhoid and small-pox of 108·3 and 107·6 respectively. See also 'Med. Times and Gaz.,' Jan. 1st, 1870. Andral asserts that the fibrin never exceeds 10 parts per 1,000 without the temperature exceeding 104°.

[2] See Cases 11 and 12 of Class IV. Appendix D.

[3] Dickinson's 'Pathology and Treatment of Albuminuria,' p. 231.

Pneumonia a general affection having its local expression in inflammation,

We are set then to seek, as offering some analogy to pneumonia, for some affection general as well as local, which shall exhibit, together with this phenomenon of increasing hyperinosis, a special tendency of its own—sometimes fulfilled and sometimes only threatened—for some particular seat of inflammation. I think we find it in rheumatic fever.

resembling in this respect acute rheumatism.

I am aware that pneumonia is quite different in its general deportment from rheumatic fever. The course of the one is even and regular, of the other broken and erratic. The one recurs, the other does not. To liken them would betray a total disregard of the daily teaching of the wards. Nevertheless, for the phenomena of the one there are analogous or corresponding phenomena in the other. There is a parallelism between the two which is not affected by their distance apart. Thus, in acute rheumatism we have a pyrexia sometimes preceding and sometimes coincident with the joint inflammation; we have an irregular and indefinite course of fever, often out of harmony with the progress of the local affection,[1] with a special liability to inflame on the part of a certain membrane—the pericardium. In pneumonia we have a sudden pyrexia, sometimes preceding and sometimes accompanying the local affection, and in many respects independent of it. Both rheumatic fever and pneumonic fever are recognisable of themselves from their intrinsic qualities, and while each has its own selected point of attack—the pericardium in the one case, the lung and pleura in the other—the stress of the malady is not necessarily expressed in that way. Acute rheumatism may give rise to pleurisy in place of pericarditis; the general signs of pneumonia may fail of their promise so far as the lung itself is concerned.[1]

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[1] The irregularity of the rheumatic pyrexia is an accident due to its local manifestations, the inflamed joints set up a pyrexia of their own. As the persistence of pleurisy and the pyrexia due to it in its individual character may prolong and disfigure the course of pneumonia pyrexia, so, but to a greater extent and more often, a number of separate seats of inflammation in the joints will prolong and disfigure the course of rheumatic fever. But what is an exception in pneumonia is a rule in rheumatic fever. We can trace the natural course of the one pyrexia, but hardly of the other.

Compatibility of rheumatic fever with pneumonia.

And further, although these diseases display a marked individuality, yet their pathological relations are not dissimilar. Both select their subjects of about the same age, and the same mode of death—that by embolism—may suddenly intervene in the course of either. The presence of the one affection does not prevent the intrusion of the other. In the full tide of rheumatic fever there may occur, as we have seen, a quite typical pneumonia. Such a combination is the more to be insisted on because its significance is apt to be underrated. Pneumonia is constantly spoken of as "complicating" all sorts of diseases. When therefore it is asserted to be one of the most fatal events of acute rheumatism, the phrase may be taken as mere common form. Yet, for the particular set of phenomena which, rightly or wrongly, we are here discussing under the name 'pneumonia,' to interpose in the course of another affection is a notable occurrence, the possibility of which in the present case must be taken to indicate a certain kindred between two affections whose points of difference are nevertheless clear and unmistakable.[2]

Pericarditis common to both.

The same relationship may seem to receive further and independent support from the pathology of pericarditis.

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[1] As for example in Andral's case, quoted in a note to p. 33.

[2] It might be urged conversely that acute rheumatism sometimes interrupts pneumonia. Grisolle reports four cases of the latter disease in which an inflammatory condition of the joints occurred. I have no personal knowledge to confirm such a view. The rheumatic character of these examples may perhaps be open to doubt. It is best to leave them out of the case.

This affection, as we have seen, both precedes and attends pneumonia, and, judging from fatal cases,[1] must be looked upon as intimately associated with it. Now it is to the pericardium, notoriously, that the rheumatic poison is chiefly directed, so that, in their several degrees, pneumonia and rheumatic fever exhibit a common tendency in respect of this particular membrane.

Relation of erysipelas.

Does true pneumonia arise in any other pathological connection? It will be remembered that Trousseau,[2] upon scantier grounds perhaps than would be accepted from a smaller authority, has discovered a strict analogy between erysipelas and that form of pneumonia which, commencing at a small spot, rapidly spreads over the whole lung. Now, whether or not it be true that pneumonia having this character is properly likened to erysipelas, certainly cases are met with where this latter disease follows so closely upon pneumonia (whether or not of the kind described by Trousseau) as to interrupt recovery, and such attacks are sometimes recurrent.[3] The connection is rare and ill-defined, however, and little can be deduced from it as to the nature of either affection, but the two conditions are not incompatible.

Renal disease favouring pneumonia

With diseases of the kidneys the connection is more obvious. There is ample post-mortem evidence of the concurrence of pneumonia with acute tubal nephritis and with amyloid degeneration, especially the latter. Speaking of it, Dr. Dickinson writes: 'There is a marked tendency to inflammation, showing itself most often in the lungs as pneumonia, next in order of frequency as pleurisy. Pneumonia in particular is more frequent in grown persons suffering from this disease than in either of the other forms of renal malady. Bronchitis can hardly be regarded as a concomitant of the disorder. There is, as with other forms of renal disease, a tendency to erysipelas or to inflammation of the cellular tissue.' 'It is probable,' the same author observes (although the evidence upon the subject would appear to be extremely scanty), 'that both with "depurative kidney" and tubal nephritis the fibrin yielded by the blood is largely in excess of health.'

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[1] See p. 78. [2] Trousseau, 'Clinical Lectures,' iii. p. 353.

[3] In 1864 a man of 60 was admitted into St. George's Hospital with symptoms at first taken for fever. On the third day he exhibited pneumonia, having its seat at the upper part of the right lung. On the way to recovery facial erysipelas set in. Getting well of this he returned, some time later, with a second attack of it. Grisolle quotes from Serres an instance of several attacks of pneumonia in the same patient, each ending with erysipelas. Dr. Wilson Fox has met with erysipelas once in pneumonia, three days after the decline of pyrexia. Fox, *loc. cit.* p. 652.

owing to interference with elimination.

In this liability to suffer pneumonia from impairment in the excreting power of the kidney, may we not see, brought about through obvious structural defect, the very same mischief which is provoked equally by external and accidental causes? There are certain essential factors whose agency in the production of this disease is not altogether beyond scrutiny. These are to be discerned both in the subjects of the affection and the circumstances of the particular attack. In the individual there is the association with serous inflammation and fibrinous exudation; in the circumstances there is some defect or impediment, temporary or permanent, sudden or gradual, in the apparatus of elimination. The blood is hyperinotic, or prone to become so, while certain elements of secretion are retained in the system. Let there now arise the accident of exposure to cold or wet, or both, let the man so circumstanced get drunk and fall asleep in a wet ditch, or, being ill-fed and ill-clothed, let him be placed in a draughty house or exposed to a cutting east wind, and a sudden outbreak of pneumonia in its most typical form is, as we know, the very frequent result. Nay, these external circumstances may be so well combined as of themselves to excite pneumonia in so large a number of vulnerable persons that the disease shall seem for the time epidemic.

Thus, although neither the state of body which immediately precedes pneumonia nor the external causes which, with the existence of that state, will suffice to excite it, can be explicitly determined, it is yet possible to surround it with conditions, and mark out roughly the area within which the operation of the disease is limited. The sort of subject for it and the sort of cause for it we know approximately, yet not so accurately as to be able to predict when the two are seen together that pneumonia will certainly ensue.

Its place among diseases.

Pneumonia thus occupies a middle place between the specific fevers so called, and the local inflammations, and has something in common with both. It falls short of the one by wanting the material of infection and a period of incubation, it exceeds the other by possessing the character of orderly pyrexia and definite duration. It is not a fever which apart from any fixed seat pervades the system generally, like typhus, and, in default of definite lesion, is laid to the charge of the blood, neither is it a mere pyrexia, the immediate response to a local inflammation. For its full recognition (I would not say for its essential existence) lung inflammation is necessary, but the presence of such inflammation by no means implies or secures the presence of pneumonia. At one time it will present, almost exclusively, the characters of a local disease, at another, chiefly those of a specific poison. By its aspect, its duration, and concomitant lesions, as well as by the special character it acquires in certain epidemic constitutions, it would seem most like fever; in its actual histology, the class and circumstances of persons amongst whom it obtains, and the absence of any extraneous material cause, it is most like an 'idiopathic' inflammation. And this position of isolation between distinct classes of disease is further secured to pneumonia by the fact that, in the best instances of the disease, the lung itself does not actively participate in the changes that occur within it, that in yielding directly from the blood a material destined to be removed from the system without injury to the organ that receives it, the pneumonic process is in fact special and *sui generis.* As such let it be judged of by itself, less by comparison and analogy, which may be forced and misleading, than by endeavouring to trace from the pattern of nature its own proper history in various circumstances, careless whether in the delineation we are producing a picture in harmony with preconceived notions of what the thing ought to be.

CHAPTER XIII.

PNEUMONIA IN ITS RELATION TO PHTHISIS AND TUBERCLE.

Pneumonia in its relation to phthisis—The processes of phthisis contrasted with those of pneumonia—Pneumonia does not modify the course of phthisis—Consolidated lung in association with phthisis distinguishable from pneumonia—Destructive pneumonia in comparison with acute phthisis—Acute tuberculosis in its relation to pneumonia—The abiding products of pneumonia may eventually give rise to tuberculosis—Summary conclusions.

It is beyond the scope of this treatise to enter upon the subject of tubercle or of the various degenerations of the lung included under the term phthisis. Yet, as has been shown, the morbid anatomy of pneumonia so blends with that of phthisis, and even of tubercle, that it becomes necessary to an adequate account of the affection we are concerned with, to consider shortly the nature of this relationship. In so doing I do not attempt any description of phthisis or tubercle, or propose to advance any facts in regard to either which are not already admitted.

Phthisis distinguished by textural degeneration.

Of true pneumonia it has been shown to be one of the characteristics that its entire clinical history is included within ten days or a fortnight. The exudation from the pulmonary blood-vessels, which is its cardinal phenomenon, involves no structural change or permanent injury. Although the air-sacs over a large area become uniformly filled with a plastic material destined to undergo changes of its own, they are not themselves changed; the lung that has suffered pneumonia is undistinguishable from its fellow that has escaped.

Now, in phthisis of whatever kind, there is, to begin with, an essential difference from pneumonia in this respect, that the structure of the lung is early implicated. Along with the alveolar catarrh degenerative changes set in, and the injury done to the alveolar walls by these means is irremediable.

It is foreign to our subject to trace the steps in this degeneration, and its final issue in caseation or fibroid conversion. Sometimes a new small-celled growth infiltrating the alveolar walls arrests the capillary circulation and puts a term to the active life of that portion of lung, or the same end is accomplished by the pressure from within of imprisoned catarrhal products. Sometimes, by means of a fibroid development in the alveolar walls the lung becomes indurated and contracted. It is enough to notice that in either case the histological phenomena in their course and in their result are different from anything to be observed in pneumonia.

Assumed to be the consequence of pneumonia.

By an unfortunate nomenclature, however, the term ‘chronic pneumonia’ is made use of to indicate not pneumonia itself or any stage of it, but a fibroid or corpuscular change of the vesicular structure, which, by a bold assumption, is claimed to be the direct result of that affection. The precise manner of these changes, which terminate either in caseation or induration, it is not permitted us to see. Their progress during life is only doubtfully marked at isolated periods, not by the symptoms of pneumonia but by those of bronchial catarrh. If in that earliest attack of bronchitis in a phthisical subject, which is recalled afterwards as fixing the probable date of the earliest structural change, the lung could be actually seen, we might say whether the mischief commences as an intra-alveolar cell-growth or in the substance of the alveolar walls

Wanting that evidence, any positive assertion as to its mode of origin savours of mere dogmatism, and, like some other points in pathology incapable of demonstration, depends very much for its support upon the emphasis with which it is stated.

We have already had occasion to observe upon the wide relations of alveolar catarrh. We can say generally that, wherever there is morbid change involving the alveolar structure, there *it* is likely to be. But the converse would not be true. The filling of the alveoli with catarrhal products does not of itself endanger the alveolar walls. In that process which implicates them most—in pneumonia—they do not suffer at all.

Phthisis and pneumonia independent of each other.

The relationship between phthisis and pneumonia is accidental. The processes of tissue degradation and fibroid growth which constitute phthisis do not produce pneumonia, nor are they, except rarely and doubtfully, influenced by that disease. Thus pneumonia, occurring to the subjects of phthisis, will run its ordinary course to its ordinary end without affecting, so far as can be seen, the chronic ailment; while phthisical persons, although they have a morbid liability to ordinary catarrh, are not more exposed (are, I believe, as a fact, less exposed) to pneumonia than their neighbours.[1]

Congested or consolidated lung in association with phthisis.

It may be urged, on the other side, that in a large majority of post-mortem examinations after death from phthisis, pneumonia is noticed along with the caseation or the fibroid induration. Certain parts of the lungs remote from these will be 'red and congested,' or in parts 'hepatised,' or while one lung is riddled with vomicæ, its fellow will be in 'the first stage of pneumonia.' This is the customary language of such accounts, insomuch that a perusal of them would lead to the conclusion that pneumonia in some form or other, so far from being an accident, was an invariable concomitant of phthisis. We must note, however, the exact manner of this association.

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[1] When undoubted pneumonia attacks the subjects of chronic phthisis, the acute disease both as to its seat and general progress is quite independent of the other. Both Louis and Walshe state that the mean duration of pneumonia in phthisical subjects is less than when occurring in sound lungs. 'Some of the most marked examples of rapid resolution I have met with,' says Dr. Walshe, 'were in phthisical persons.' It may be added that the subsequent course of phthisis is no way altered by an intercurrent attack of pneumonia.

Its character.

In the most chronic forms of phthisis, such even as have maintained the apyrexial character up to the hour of death, the process of caseation will be found in various stages of progress. There is the excavated vomica, where the work may be said to be finished, and there are ill-defined patches of carnified or solid lung, densest in the centre and shading down insensibly into natural tissue, where it cannot with certainty be said to have begun. These latter are the appearances to which the phrases I have quoted refer, and they are described as 'the first' or 'the second' stage of pneumonia, according to their degree of density, and in remembrance of what takes place in the acute disease. Yet they represent, not pneumonia, but the several steps in a process which is essentially slow and lingering. During the lifetime of the patient the more recent of these spots have escaped detection altogether. Even to the eye they seldom have the character of pneumonic hepatisation. They are not granular in fracture, nor do they exhibit under the microscope the material of a solid exudation filling air-sacs whose proper structure is unaltered. Sometimes, indeed, this structure is so converted as to bring it within the category of fibroid lung already described.

Indurated lung, it is true, will be found sometimes apparently in such near association with pneumonia as to make it seem an overstrictness to question the unity of origin of

Indurated lung along with hepatisation.

the two. Thus in the midst of engorged lung, a portion will be disclosed in a state often described as 'red induration.' This appearance may occupy exactly the situation of ordinary hepatisation, and like it be accompanied by membranous lymph. Moreover, the colours of red and grey may be so intermixed as to suggest the successive stages in the pneumonic process, differing only in consistence, while further, this indurated lung, as if following the usual course of destructive pneumonia, breaks up at length into cavities, only by a chronic instead of an acute process.

Distinction between the two.

Yet neither on clinical or pathological grounds can such a relationship be admitted. Not only do we fail to observe in these cases the symptoms of pneumonia; there is no real likeness after death. The history of the patients is the history of phthisis; wasting, hectic, often diarrhœa, irregular febrile accessions, intervals of imperfect health, expectoration bronchial or puriform, sometimes the earliest sign hæmoptysis. The symptoms have seldom if ever had their commencement in an undoubted attack of acute pneumonia. After death the indurated lung, which is often contracted, is either firmly adherent or surrounded by a tough membrane. Moreover, the process of softening by means of which excavations are at length formed, proceeds at separate points, each distinctly bounded; the intervening lung is still tough and, it may be, almost cartilaginous. As the disease advances, an investing tissue surrounds and isolates these softened portions, which thereupon become gangrenous.

Thus the pneumonic lung, and this form of indurated lung, instead of being similar are in truth as widely separated as is well possible for two conditions befalling the same organ. With the acute affection the lung expands and softens; with the chronic it contracts and hardens. With pneumonia the purulent conversion is irregularly spread; with induration separate foci become the seat of a destruction which is abruptly marked off and isolated. Induration tends to gangrene, pneumonia does not.[1]

Pneumonia as a destructive lung affection contrasted with destructive phthisis,

But, it may be said, if all the destructive forms of lung disease are to be classed as phthisis, pneumonia must needs be included, whenever it assumes that character. Fettered by such a definition, there is always phthisis where pneumonia destroys the lung by purulent infiltration. The dilemma, while it exposes the imperfection of our nomenclature, illustrates also the essential difference between the disease we are discussing and all those conditions commonly classed as phthisis. You shall have two such patients side by side, the pneumonic and the phthisical. Both will have pyrexia, for both are undergoing rapid transformation of some portion of the lung. Yet in other respects than those in which, owing to the implication of the same organ, the patients must needs suffer alike, the difference between them is apparent and in some instances very striking.

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[1] See some cases of the kind reported by Dr. Morehead, 'Diseases of India,' vol. ii. p. 338 *et seq.* The features of similarity and difference between the two conditions from the clinical point of view are sometimes strikingly exhibited. My friend Dr. Bagshawe of Hastings lately called my attention to an instance of the kind in a patient of his own—a gentleman a little past 50. Here the whole of one lung was dull to percussion, the respiration markedly tubular and vocal resonance greatly in excess. The affected side, however, measured $\frac{3}{4}$ of an inch less than the other, and the voice sound wanted the brassy ring which one expects in hepatisation. The patient's condition was simply that of a man wanting a lung. That is to say, he was easy when at rest, but became breathless on slight exertion. The pulse and body temperature were normal, the appetite good, and digestive organs undisturbed. The patient was of healthy family, of active business habits, with no tubercular or phthisical history; but some years before he had contracted syphilis. His first notice of illness consisted in an attack of hæmoptysis, and from that time he had complained, and complained only, of impaired breathing power. Dr. Bagshawe's view coincided with my own in regarding the case as an instance of fibroid lung, probably the result of syphilis.

Since this my friend and colleague Dr. Cheadle has communicated to me the particulars of a case of fibroid lung occurring in a syphilitic patient, and very similar in its section to that figured on page 136. From the physical signs it was put down during life as pneumonia.

in symptoms

Both will be pyrexial, but the profuse sweating of the phthisical patient will not be seen in the other; both will have dyspnœa, but they will take it differently. The subject of pneumonia exhibits listlessness and mental obtuseness like that of continued fever, or he is disturbed by active delirium; at any rate, the disease that possesses him alters both his aspect and mind. The other, though not without night-wandering, will be quite unchanged in mental respects. His face, pinched and drawn, will betray an anxiety as to his future he takes pains to conceal. To the last he will maintain a keen interest in passing events, with that confident expectation of recovery which so remarkably distinguishes the victims of pulmonary consumption.

and morbid anatomy.

With such obvious differences the auscultatory signs of the two may be almost identical, for in both portions of lung have become solid. Yet if these organs be examined after death in the two cases, certain points of difference will appear. The pneumonic lung will exhibit only the remains of lung structure; the alveoli are broken down and their walls exist only as scattered fragments amid a multitude of pus corpuscles which, from this merging together of separate air-sacs, come almost alone into view. The state of the caseating lung will be similar but not the same. There destruction will be less, but a special character of the disease will appear in the changes which have befallen still existing lung structure. The vesicular arrangement is still distinct, although it is but the skeleton that remains. The alveolar walls are indeed broken down at certain points, so that a number of contiguous spaces are merged into one; but besides this they are themselves infiltrated with a small cell growth, which is more or less distinct from the material they enclose. And along with this appearance, though in very variable degree, these alveolar walls often exhibit a fibroid conversion, by the intervention of which, sometimes, the active spread of destruction in certain directions is abruptly stopped and limited.

It seems probable, therefore, (although at the latest stage a common ruin may obliterate all distinction,) that purulent pneumonia and acute phthisis[1] are as separate in their anatomical history and progress as they undoubtedly are in their general symptoms and associated pathology.

Pneumonia in relation to acute tuberculosis.

With tuberculosis the case is similar. The relation between it and pneumonia is remote and uncertain. Tubercle does not give rise to pneumonia; pneumonia has no appreciable effect in exciting tubercle. The grey granulation (inseparable as it is from other modes of tubercular infiltration) is nevertheless an independent structure, anterior as to its birth to the alveolar catarrh which *post mortem* is so constantly found associated with it.[1]

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[1] A comparison like the above between individuals dying, it may be, at different periods of their respective diseases, is, I am aware, open to exception. I am but quoting what I have seen. The point may be put more absolutely thus :—In acute phthisis the caseation commences at separate districts in lobules or groups of lobules. Each of these becomes the seat of a separate inflammation, having its own rate of progress tending in every case to the ultimate conversion of the central portion into pus. On post-mortem examination therefore, the section of such a lung (probably at its apex) presents a riddled or honeycombed appearance. In the most acute cases there is no trace of fibroid development. Every little cavity evacuated of its creamy contents shows itself as a mere ragged hole excavated in the bare substance of the lung. The pulmonary tissue intervening between these suppurating centres may next become engorged or hepatised. If sufficient time be allowed it may itself become infiltrated with pus and ready to break down. Yet this general implication is always secondary to that of the separate centres. There was a time when these were alone affected. It is not as with pneumonia lobar hepatisation first, and next the breaking down of the lung at several points ; it is the reverse of this. See Case 6, Class II., Case 11, Class V. Append. D. See also Dr. Williams' 'Pulmonary Consumption,' p. 217.

Pathology of tubercle.

In acute tuberculosis, there is strong reason to suppose, we have neither an exudation nor a new growth, but the hyperplasia of certain normal elements, by means of which the lung, in common with other structures, becomes studded with grey, semi-transparent granulations. The true granulation has its seat in the connective tissue, while the neighbouring alveoli, distorted and blocked by the projection of these bodies, are apt to exhibit proliferation. A certain number of alveoli may thus be obliterated, but their obliteration is accidental, and not, as in catarrhal inflammation, the main feature of the morbid change.[2]

And accordingly, acute tuberculosis is seldom recognised at the bed-side by any local inflammatory action on the part of the lung. It is judged of by general rather than special signs. With individuals of a certain disposition or diathesis, great nervous depression and high fever, not otherwise accountable, will give rise to a suspicion of tuberculosis. Such cases are often mistaken for typhoid fever, and even when that affection has been excluded the diagnosis rests at last rather upon negative than positive grounds.

The cause of acute tuberculosis as it concerns the lung.

And while it is impossible during life to obtain direct evidence of acute tuberculosis, it is to be observed that, in the system of exclusion which finally leads to a correct inference, pneumonia finds a place. We get a step nearer to a conclusion by the observation that the disease is *not* pneumonia. It is a part of the ordinary text-book instruction to point out how in its physical course acute pneumonia differs from acute tuberculosis, how in the one the crepitant râle gives place to the sounds indicative of consolidation, while in the other this râle persists, so that days after its first appearance 'the signs of hepatisation are not one iota more obvious than the previous day.'[1] This distinction, which clinical observation witnesses, morbid anatomy fully confirms, yet not without room for error which it may be worth while to notice.

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[1] Professor Klein, in his work just published, 'The Anatomy of the Lymphatic System,' Part II., affirms that alveolar catarrh is at the very beginning of the tubercular process.

[2] See Rindfleisch, 'Pathologieal Histology,' ii. 39.

The grey granulation, as has been said, hardly exists apart from catarrhal cell development on the part of the surrounding alveoli. Within this area it is possible to select particular spots where these elements are undistinguishable from those of ordinary pneumonia. Now along with this catarrh, which is limited to the neighbourhood of the granulations, the entire lung, thus suddenly beset at many points, is apt to become hyperæmic. We have thus, at once, congestion like that of the first stage of pneumonia, and here and there, in isolated spots, sparsely or thickly scattered, the products of inflammation grouped round the several granulations. Yet the likeness to pneumonia, such as it is, is not long maintained. The congestion is sufficiently distinguished by its history. Its origin, duration, and ultimate destiny are all different. The engorgement of pneumonia is but the first step towards the exudation which is the great event of the disease, while this secondary congestion is stationary and remains unchanged as long as the affection to which it owes its birth. The inflammatory products around the several granulations we have seen already, and learnt to recognise, as the constant accompaniment of a localised pulmonary disturbance.

The hyperæmia of tubercular lung to be distinguished from pneumonia.

In the next place, it is to be observed of pneumonia that it has no appreciable effect in exciting tubercle. It is not, as we might suppose, that a susceptible or vulnerable lung is at once excited to this form of hyperplasia by the stimulus which pneumonia supplies. On the contrary, we see children whose family and personal history point most plainly to acute tuberculosis, and who ultimately may even die of that disease, undergo pneumonia with no further detriment and no more prolonged illness than others. We see them die of it, as others die, the tubercle scattered throughout the inflamed lung but not actively concerned in the morbid process.

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[1] Walshe, 'Dis e Lungs,' 547.

Both phthisis and tubercle share in the histological elements of pneumonia.

Regarding pneumonia, then, in its anatomical relations, not as a whole but in bits, not as a disease but as a collection of histological phenomena which may be variously associated, certain of its elements are to be recognised in the proliferative and other changes which accompany caseation and acute tuberculosis. But viewing it in its integrity, in the light of its history and mode of progress, these same elements occupy a special place and contribute to a special end. Their character is determined by their conduct. The same histological elements in the one place represent the final result of a local disturbance, in the other a single stage in a progressive and orderly series of changes.

Pneumonia as the starting point of phthisis and tubercle.

There remains still the question whether true pneumonia unusually protracted may not at last degenerate into phthisis, and by that avenue eventually excite tubercle. That pneumonia does so end sometimes, there is strong reason to suspect. Nevertheless, in examining the particulars of cases of phthisis originating, as is asserted, in inflammation, it is quite rare to find a clearly defined, undoubted attack of pneumonia as the starting-point.

Probably with most forms of chronic phthisis the earliest morbid changes concern the alveoli alone. Certainly the fact is that the history of such cases reaches back to a period of ill-health antecedent to any complaint of the nature of acute inflammation. It is not until the phthisical affection is fully declared that sundry of the patient's antececedents are arbitrarily selected to father it. When the individual history is thus ransacked, undue prominence may be given to certain events of his life, and the account transmitted for our instruction comes through a distorting medium. Yet even so, it is seldom that chronic phthisis can be traced right up to its source in any of those catalogued disorders which are credited beforehand with the power to produce it.[1] The inflammation that is most associated with phthisis is bronchitis, but it is related to it much more as an intercurrent affection than as the initial one, and much oftener in the form of bronchial catarrh than as an acute inflammatory attack.

Pneumonia is sometimes set down as the starting-point of phthisis because in auscultation the physical signs resemble those of hepatisation or inflammatory engorgement, or both. Carried away by that observation, the antecedent history is disregarded. There is a 'latent form' of pneumonia always at hand to do duty on such occasions. Let the patient present the appropriate local signs, and let his illness be recent, and we arrive at once at that diagnosis. The pneumonia so established soon obtains by its persistence the further name of 'chronic,' and upon its termination in death is supposed to afford a suitable illustration of 'pneumonic phthisis.'

Pneumonia giving place to phthisis, which may proceed to caseation or fibroid degeneration,

I do not however deny that true lobar pneumonia does sometimes end in phthisis. Only the event is so uncommon, and, when observed, the connection is so indistinct and uncertain, as to make the term pneumonic phthisis essentially misleading. Thus, in rare instances—usually with persons of phthisical history—pneumonia will occur in the usual way (or more often its physical signs will occur) in one whose health has for some time been impaired.[2] Long after the acute symptoms have disappeared, the local products of the disease will remain, keeping a portion of the lung solid. The patient meanwhile either wastes, and soon begins to exhibit the characteristic symptoms of phthisis, or else (and this is less uncommon) he continues for a considerable time, in spite of the local defect, in fair though delicate health.

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[1] If it were required, without afterthought, to name the symptom of all others which most often stands at the very beginning of the phthisical history, I suppose it would be hæmoptysis.

[2] A reference to the valuable records of Dr. Williams's own cases in his work on Pulmonary Consumption will fully bear out the statement of the text, both as to the ill-marked and uncertain character of these antecedent inflammatory attacks, and as to the probability in

ending in tubercular infection or becoming obsolete.

Results so opposite find their explanation in pathology. In the one case the accumulated products of inflammation undergo and induce degenerative changes which in their turn, by an infective process, give origin to true tubercle ; in the other these same products are attended by an increase of the fibrous tissue of the lung, with fibroid development or conversion of the alveolar walls by means of which, temporarily or finally, the seat of disease is constricted and hedged round, and its extent of harm limited to its own neighbourhood.

Caseation, latent for a while, giving origin at length to tubercle.

Sometimes, for reasons that are rather conjectured than known—exposure, neglect, insufficient nourishment, or what not—a patient with no flaw in his history will exhibit in turn both these phenomena. He will recover, that is, his health, but not the use of his lung ; and after an interval, longer or shorter, his local disease will assume a new activity, and he will finally perish by acute tuberculosis. In such cases while to outward appearance and his own feeling the patient is well or nearly well, when he is not losing but gaining flesh, the stethoscope will reveal, in all their completeness, the physical signs of consolidated lung just as these exist at the very height of acute pneumonia. This, if anything, may be loosely called chronic pneumonia, and the termination of such cases is full of interest. I may allude

many instances that the actual commencement of phthisical disease, although not formally recognised, was in fact anterior to the acute symptoms. See especially Chap. xviii., 'Cases of consumption originating in inflammation.'

very briefly to one under my own observation for several years, which, although unfortunately incomplete, from the absence of post-mortem examination, affords, nevertheless, a sufficient illustration of my point.

Case of pneumonia fatal by tuberculosis after several years.

In the spring of 1863 a young woman, Elizabeth W., aged 18, presented herself at the Chelsea Dispensary complaining of cough with some mucous spitting, the remains of an acute illness for which she had been treated at her own home. The girl was plump and florid, and had the aspect of health. Respiration was obviously hurried, however, and the pulse frequent. The parents were healthy, and on both sides the family history was remarkably free from phthisis. On making examination of the girl's chest, the lower two-thirds of the left thorax was dull to percussion with increased vocal resonance, highly tubular respiration, and, at the base, a crackling crepitus only a little coarser than that of commencing pneumonia. To be brief, this patient reappeared from time to time at varying intervals spreading over more than three years, with precisely the same physical signs and with general symptoms never very grave, and confined for the most part to troublesome cough with some bronchial spitting. Although her occupation as a dressmaker took her into the open air in all weathers, she was not noticeably affected by the seasons; she did not lose, but gained flesh.

Having lost sight of this girl for some years, from resigning the appointment of physician to the Dispensary, I was again asked by the mother to see her in the autumn of 1866. She was now much wasted, the expectoration was mucopurulent and nummellated, whilst the sounds on the affected side (which was still, as before, dull to percussion) indicated that in places the lung was beginning to break down. I now learnt that the patient's general condition had altered, and she had been losing flesh for some weeks. She suffered from night-sweats, and had had several attacks of hæmoptysis, for which she had been an in-patient at the Brompton Consumption Hospital. Her condition was now in no respect different from one of tubercular phthisis in an advanced stage. So the girl died, and owing to an unfortunate mistake no post-mortem examination was obtained. But the point of interest in the case refers to the earlier part of it. The physical signs at the close clearly indicated that solid lung, long quiescent, began at length to break down at several points, while coincidently with this the patient, who had hitherto only suffered from the local affection (her general health remaining good), puts on all the symptoms of acute phthisis and is quickly destroyed.

Resembling this case in its earlier history, and but too likely to resemble it in the sequel, is that of a child lately discharged from the Hospital for Sick Children.[1]

Case in which the products of inflammation remained.

Sophia T., on admission to the Hospital (November 27, 1873), was six years and a half old. Her uncle had died of phthisis. She had been ill a month with cough and pain in the chest, spitting thick, uncoloured phlegm. Latterly the symptoms had increased, and some diarrhœa had been noticed. The child was now acutely febrile, having a pulse of 182, and temperature 104·8°. The whole of the left side posteriorly was dull to percussion with increased vocal fremitus, and over the lower part, but most markedly immediately below the scapular spine, respiration was intensely tubular. There were moist crackling sounds over both lungs. The notes of this child's progress, extending as they do over several months, are not easy to summarise. It may suffice to say that such progress was very uneven and erratic, the temperature rising and falling irregularly, while the child's appetite, and weight, and general condition were not less variable. She had profuse sweating at times, the sputa were muco-purulent, with sometimes a little blood. These changes in the child's health seemed to depend on, or rather they corresponded with, intercurrent attacks of bronchitis to which she was liable. The lung remaining solid was yet free from time to time of all added sound, and in such intervals the fever would disappear, the appetite become voracious, and the child rapidly gain in flesh. Presently, both the diseased and the sound lung would yield small crepitant râles, the temperature would again rise, and the general health decline. Upon measurement it appeared there was some contraction of the left or affected side in all its diameters. The variations in the child's weight indicate in some degree the course of the complaint. On admission she weighed 2 stone 3½ pounds; this weight increased with slight oscillations to 2 stone 7 pounds on April 27, and on May 28, the date of her transfer to Highgate, it was still the same.

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[1] This girl was admitted under the care of my friend and colleague Dr. Cheadle and afterwards transferred to the Convalescent Branch of the Sick Children's Hospital at Highgate, where she came under my own charge. Dr. Cheadle, in furnishing me with the particulars of the child's early illness, is kind enough to consent to my quoting the case here.

Of the progress at Highgate little need be said: in her bronchial attacks, which were still frequent, she suffered less general disturbance than formerly; there was neither hæmoptysis nor any form of spitting. As regards the physical signs, while the tubular breathing remained, the dulness of the left thorax diminished in extent and degree, and, from the alteration in voice-sound and vocal fremitus which occurred, the small area of absolute dulness still remaining at the base of this lung seemed due to pleural fluid. The physical signs thus indicated that consolidation was now confined to the central portion of the lung. So the child left the hospital, after a residence of eleven months, to outward appearance well, but with the material results of a presumed pneumonia still present and a ready source of danger.[1]

Points of similarity.

Now mark the similarity between this case and the last. Both are preluded by a period of feeble health. In both the general symptoms undergo distinct and frequent intermissions during which the continued presence of disease is only discoverable by the stethoscope. In both the physical signs indicate early consolidation of the lung, a condition which (wanting in some of the characters of pneumonia) persists when all pyrexia has disappeared, and the patient is growing fat and about her ordinary work or play. If this be phthisis it singularly belies its name. But observe again, in the fatal instance, the rapid change in the character of the complaint: wasting, hæmoptysis, night-sweats appear together, and, along with these, physical signs of disorganisation on the part of the lung.

The future of such patients.

Cases of a similar kind are not uncommon, but death is delayed till long after they have ceased to be distinctive, anatomically. The morbid elements which the lung presents—fibroid increase, caseous softening, grey granulation —do not by their mere presence discover the relationship which they hold to one another or indicate the earlier stages of the disease. There is much reason to concur in the belief that the retention of catarrhal products is amongst the first of these—retention due, I cannot suppose to an accident or any circumstance within the control of drugs or climate, but to something or other in the vital condition of the patient. Governed by this the process of deteriorative metamorphosis tends at one time to fibroid hyperplasia and conversion, at another to fatty or granular degeneration.

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[1] As regards its relation to pneumonia, I would ask the reader to compare this case with that described on p. 118. See also a case of recovery in Dr. Williams' 'Pulmonary Consumption,' Case 57, p. 204.

With the former and more favourable event, the implicated portion of lung for a while undergoes a fibroid change with contraction, rather than a process of softening with subsequent evacuation and the formation of irregularly diffused cavities. There is fibrosis in place of 'crude tubercle,' or, more truly, the original catarrhal elements undergo a process of caseation of their own (the alveolar walls being implicated here and there), while the disorganisation which that change implies is, to a greater or less extent, limited, and, as it were, isolated by a concomitant fibroid development.

Unremoved products becoming isolated and obsolete,

Yet caseation, however limited and shut in, is always a possible source of infection; its presence, almost harmlessly for a length of time in some individuals, is accounted for, hypothetically, upon the supposition that such persons are difficult to infect, that the adenoid structure of their lungs is slow to respond to the irritation to which it is subjected. With them two things are possible. One, the eventual obliteration by fibroid increase and contraction of the caseous spot, and, along with its obsolescence, recovery; the other, caseation advancing in excess, and in spite of the concurrent fibroid change, at last by its persistence infecting these adenoid tufts, and thus eventually developing tubercle. The particular result in any given case is determined not only by the constitution and proclivities of the individual, but by many accidents of life which either improve or deprave vitality. Of the predisposed the fate is not doubtful; with the rest it is possibly within our own power to some extent to control the result.

or else the source of tubercular infection.

And the advent of tubercular infection (as with the girl first alluded to) is not disguised—wasting, hectic, hæmoptysis, altered sputa, together with the physical signs of cavities forming in the lungs, all attest that a new disease has been imported. At the outset the affection is a local one, consistent with plumpness and fair health; presently it

The clinical correspond with the pathological phenomena.

becomes general and puts on for the first time the features from which it takes its name.

Summary conclusions.

The subject, however, cannot be pursued further within the limits to which I at first proposed to confine it. The statements of this chapter, dogmatically expressed, may be summed up in a few words thus:—

1. The processes of phthisis necessarily involve the proper structure of the lung; those of pneumonia only occasionally involve it and then secondarily.

2. Yet phthisis and pneumonia are allied in this respect, that alveolar catarrh is associated with both. Differently, however, in the two cases. In phthisis the catarrhal elements are the agents in a process of disorganisation which their presence assists. In pneumonia this same catarrh is provoked by the occurrence of an exudation from the formed elements of which it is not always distinguishable. Its part and lot are one with this exudation, which, without any direct participation on the part of the lung, liquefies or undergoes purulent or granular conversion and eventually disappears.[1]

3. If under the term phthisis we are to comprehend all destructive lung processes whatever, it is manifest that certain forms of pneumonia must be included. Yet the contrast which destructive pneumonia presents, both clinically and anatomically, to every recognised form of phthisis is so conspicuous that the definition needs to be so far amended.

4. In exceptional cases, from causes which are mainly conjectural, the material products of pneumonia may remain indefinitely, long after the acute disease has reached its term. The sequel of such cases is various. They may terminate in caseation, or in fibroid conversion with induration, or these two processes may occur together and side by

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[1] Compare 'Anatomical Characters of Pneumonia,' p. 115.

side, or either may at any time be replaced by the other. With the caseation there is the further liability to tubercular infiltration; with the fibroid conversion and concurrent contraction the lung may practically recover, yet not without textural loss. Clinically it is to be observed of these exceptional cases that the antecedent health of the patients is usually such as to afford ground for suspecting the integrity of the lung at starting, while the acute attack which passes for pneumonia is commonly wanting in the typical phenomena of that affection.

5. Phthisical and tubercular subjects are not more prone to pneumonia on that account, and pneumonia occurring to such persons has no other course or termination than when happening to others.

6. It is probable, nevertheless, from clinical observation, that an attack of pneumonia may in rare instances hasten the development of acute phthisis. Yet since the first step in the process of caseation is not discoverable by any direct auscultatory sign, and since the particular attacks of pneumonia that have been credited with this influence have occurred to persons already suspected of phthisis, it is impossible to speak positively upon the subject.

7. The lungs of phthisical persons, like other lungs, may be found after death hyperæmic or consolidated from a number of causes quite apart from pneumonia. The more frequent presence of this condition in phthisis than in some other affections may be accounted for by the lingering death common in the disease, and perhaps also by the mechanical obstacles to the circulation in the lungs themselves.

CHAPTER XIV.

HISTORY AND STATISTICS OF VARIOUS MODES OF TREATMENT.

The numerical method of estimating the results of treatment—Its difficulties—Rates of mortality at various periods—Lessened mortality of the disease in the present day—Treatment by bleeding—Antimony in large doses—Practice of Laennec and Andral—The 'jugulant' method—British practice early in the century—Schism—Expectant treatment—Views of Dr. Hughes Bennett—The restorative treatment—Statistics.

THE subject of treatment—of the mode of influencing beneficially the natural course of a disease—is just now especially difficult and embarrassing. Particular drugs can no longer be recommended on the ground of usage or the authority of great names. Conflicting views as to the virtues of the materia medica are not now to be settled by the use of *à priori* arguments. It is easy to give good reasons for bad remedies. We can trust only the teaching of experience.

Difficulties of the statistical method.

Of such teaching—or of teaching that claims to be such—there is indeed no want, as we shall presently see, in the literature of pneumonia. Yet, in spite of a mass of evidence of this kind, it is doubtful whether the statistical method can ever be applied to settle the question. If it were possible indeed for the same observer to collect examples numerous enough for the comparison of like cases one against the other, and if, with that material, he could put in practice without favour or prejudice, at one period and in one set of circumstances, several modes of treatment towards as many groups of patients all similarly affected, he might possibly arrive at conclusions sufficient at least for his own guidance. But the comparison of the results of one observer with those of another, of the same disease at different periods and seasons and places, of cases treated upon a plan which is in favour with those treated upon a plan that is distrusted—in a word comparison and tabulation under the only conditions possible—can never be trustworthy or conclusive.

Even supposing that some one might be found practised enough to be reliable, yet sufficiently unbiassed to deal equally with conflicting methods, the result of his labour, however ample the field, must still be incomplete. At the most no more would be determined than the relative value of some two or three modes of treatment; not the best method would be thus arrived at, but only the comparative merits of a very few methods, no one of which could be assumed to be good.

Moreover, it is very difficult to estimate by mere narration the comparative severity of cases, so as to set them fairly one against the other. For the purpose we are speaking of comparison is hardly possible without personal observation, while it is hardly just unless the cases compared are subjected to the same external influences – happen, that is, at the same time[1] and place. Yet it needs not one but many observers, not a single occasion but a wide stretch of time, before a sufficient body of examples can be collected to form the material for general conclusions.

For such reasons as these the method by counting, which at first commends itself as the best, is in fact untrustworthy, the necessary conditions being unattainable. Every advocate of a particular remedy finds numbers on his own side. They are more easily invoked to support foregone conclusions than used for the actual discovery of truth.

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[1] The discrepancies between the mortalities at *various periods* under similar treatment and with the same physicians are remarkable. Thus Bamberger during 6 years had a mortality of 11·2 per cent. for the first three and of 18·19 for the second three. Brandes of Copenhagen found the rate vary from 5·4 to 31 per cent. in two successive years. In 1858 the mortality at St. Thomas's was 17 per cent., and in 1859 it was 5·7 per cent. Other reports might be quoted to like effect. See Appendix G. Also Dr. Peacock's 'Report on Cases of Pneumonia,' St. Thomas's Reports, vol. v., pp. 18, 19.

The numeical method applied to pneumonia.

These considerations, applicable to all diseases for which no specific remedy has been found, are of special cogency in the case of pneumonia. Owing to the peculiar nature of this disease it is often impossible, in quoted cases, to be assured, I do not say of its gravity, but even of its existence. Where this is beyond dispute there is still the necessity of classification.

The issue of pneumonia depends, for example, so intimately upon age that the comparison of different periods of life is like comparing different affections. Infants, and children, and adults, and old men have each, as regards pneumonia, a death-rate of their own. In certain subjects, as we have seen, pneumonia is especially to be dreaded, while in others a condition that is confused with it is no more than the expression of impending death. What can be the value of a statistical table in which all these things are jumbled up?[1]

There are other elements of difficulty. The details necessary to be known are more numerous and complicated and open to misconception than with other diseases. The condition we are engaged in considering, in so far as it is implied by physical signs, is itself matter of inference, not of direct observation. The phenomena to be registered must be first interpreted.

Nor, if these difficulties could be surmounted, can we suppose it possible that any rigid system of treatment formally proposed beforehand can actually be put in practice in all its integrity. Whatever the principle of treatment, it must be administered by an intelligent hand, and altered and adjusted to suit the changing circumstances of the case. If matters go well it may be nearly followed; if they go ill it will be variously modified or wholly abandoned. In this adaptation of means to ends, with a single view to save the individual life, no inflexible method can find a place. Granted, therefore, that the material and the discernment could be found for the correct grouping of cases in sufficient numbers, where shall we find the physician who would pursue without deviation a prescribed course of treatment in regard to each patient?

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[1] It is true, of course, that these sources of error tend to disappear as the numbers compared are increased, so that a great disparity in the death-rate of two large multitudes ever so mixed would tell at last in favour of one treatment rather than the other. Only, as will be seen, we never get the numbers, or anything approaching the numbers.

Character of the evidence.

And if from the kind of evidence that we might justly require we turn for a moment to the kind that is actually offered, what do we find? Some scattered tables (in no case dealing with large numbers), without classification of age, or detail of physical signs, or indication by which to judge of the severity of the disease. No more, indeed, than a statement of numbers, with the assertion that a death-rate, which to us now seems large, has been so far reduced by a particular method of blood-letting or the use of a certain drug. There is little in the nature of impartial experiment. The practice is determined beforehand, the cases are adduced in support of it.

Its general teaching.

From such material conclusions are to be drawn rather from what transpires incidentally than from what is directly affirmed. It would appear, for instance, from this evidence that since the time pneumonia was first rigidly defined up to the present day, its rate of mortality on the whole has been decreasing. It would seem, further, that of all the circumstances affecting this rate the time of life is the most potent; the old are likely to die, the young to recover. It will be found, too, as we shall see, that a decline in the mortality of pneumonia is coincident or nearly coincident with the decline or abandonment of the practice of bleeding; a fact, it must be remembered, which of itself witnesses nothing as to the relation between the disease and its supposed remedy.

Mortality less in the present day than formerly.

The practice of bleeding

With the knowledge of this lessened fatality of pneumonia it is quite certain that we shall not revert to systems of treatment which, however appropriate to their own time, consisted with a higher rate of mortality than that we observe now. The history of these old methods, therefore, need not detain us. 'Hujus morbi curatio,' said Sydenham, enforcing the doctrines of Celsus[1] and Galen, 'in repetita venesectione fere tota est.' Blood-letting was the only hope in pneumonia, and the times and manner of it were minutely described. The practice of England, however, was exceeded by Italy. Eighty years ago the elder Frank, in his public practice at Pavia, bled his patients twelve or fifteen times apiece. 'The mortality,' says Grisolle, 'was enormous.' Some years later (at the end, that is, of the last century), whether in mere revulsion from that extreme, or because pneumonia ceased to show itself with the same violence, bleeding was for the time almost abandoned. In Vienna especially it was rare for a single bleeding to be practised. Such forbearance did not last long, yet from time to time practitioners appeared (whom Laennec called 'heretics') who either abstained from bleeding altogether or employed it but sparingly.

But the extreme of bleeding was reserved for the present century. At the time when the allegation of Sydenham was already beginning to be questioned, there arose in Italy

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[1] Celsus, however, must not be represented as an indiscriminate bleeder. Speaking of pneumonia (lib. iv. cap. vii.), he says: 'Oportet si satis validæ vires sunt sanguinem mittere; sin minores cucurbitulis sine ferro præcordiis admovere.'

The school of Rasori.

a school which not only reasserted the supremacy of venesection, but combined with it an ally as effectual as itself. Remorseless bleeding is associated with the names of Rasori and Tommasini. In Parma and Bologna and Milan the pupils of these professors carried out their masters' teaching by removing from the subjects of pneumonia some ten pounds of their vital fluid. The plan was to bleed morning, noon, and evening for the first day, taking two pounds of blood, for the second day the same amount was obtained in two bleedings, the rest was got by repeating the operation daily, larger or smaller quantities being withdrawn according to circumstances.

Treatment by antimony in high doses.

With this draining of blood Rasori learnt to combine considerable doses of tartar emetic. It was he that discovered the possibility of obtaining for this drug a 'tolerance' on the part of the system, or, more truly, of the stomach. By continued use it appeared that larger and larger quantities could be swallowed and retained. That such 'tolerance' was not without sacrifice is rendered probable from the unexpected occurrence of death in some instances of not severe pneumonia after supposed cure, and in the course of convalescence. The rate of mortality by this treatment of bleeding and antimony was stated as about 10 per cent., a result loudly quoted in its favour, and which we must therefore suppose to have compared favourably with the results obtained under less vigorous treatment. It was alleged especially, though the evidence is obviously insufficient, that by this method hepatisation was prevented.

Practice of Chomel and Andral.

To this practice by routine and rule of thumb, dealing with the disease as a separate entity with little regard to the state of the patient, succeeded the wiser counsels of the French school of which Chomel and Andral were the chief expositors. These authors, fully persuaded of the propriety of blood-letting in general and of its special efficacy in pneumonia, objected nevertheless to the stated bleedings of Sydenham and to all fixed rules in reference to a practice which, as they thought, should rather be regulated by the circumstances of the particular case. By this time the teaching of Laennec had directed attention (as we may think now too exclusively) to the physical side of the disease. It was learnt that in pneumonia the lung underwent a series of changes little influenced by therapeutical measures. In the hope of mitigation and not of cure, Chomel and Andral and Grisolle bled and bled repeatedly, but only when the patient was strong and the disease new; they admitted many exceptions to that treatment, and recognised in particular certain epidemic constitutions where venesection was best dispensed with.

The jugulant method of Bouillaud.

This reasonable teaching was rudely broken in upon by the imperative assertions of Bouillaud. With a naked and unqualified directness, as certain to be popular as to be wrong, this physician, as lately as 1835,[1] put forward for universal adoption a treatment which went by the name of the strangling method, or method of bleeding 'blow upon blow.' By this plan pneumonia was to be strangled almost in its birth. Three days of blood-draining would destroy 'most pneumonias of the first degree about the third day of treatment.' It does not appear indeed that Bouillaud in Paris removed more blood or so much as did Rasori at Pavia. He was commonly contented with five pounds of that fluid, while Rasori often took ten. But while Rasori explicitly recognises the fact that the disease 'has always a necessary course,' Bouillaud regarded it as within his personal control; by his strangling method he could suddenly arrest it. The fact that in 102 cases so treated by himself from 1831 to 1834 it was not so arrested in 12, since that number died (a mortality greater than Rasori's), would seem inconsistent with that assertion. The professor insists, however, that these results were, in fact, favourable as compared with the death-rate of the period, and that the fatality was made up of cases too extensive to yield to his strangulation, or so far advanced as to kill their victims before there was time to put his method fully in practice.[1] Bouillaud's method was but an imitation of Rasori's. Its pretensions were greater, but, so far as comparison may be instituted, its performance was less. It had indeed something of anachronism. The time was past when the proposal to reduce treatment to a set of formulæ could escape criticism. Bouillaud was attacked accordingly by Grisolle, who only too laboriously and with the vigour of a contemporary denied that his 'blow upon blow' treatment either reduced the duration or diminished the fatality of the malady.

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[1] See Grisolle, *loc. cit.* p. 585.

British practice early in the present century.

Meanwhile the most trusted practitioners of our own country, though they might differ in matters of detail, were united in endorsing the statement of Sydenham that in pneumonia safety was to be sought by means of the lancet. While Bouillaud in Paris was pretending to crush it by repeated blows, Gregory[2] in Edinburgh was teaching that with bleeding and water gruel all other help might be dispensed with. M. Louis indeed had already expressed doubts as to whether the efficacy of bleeding were not confined to an early period of the disease, but his opinion was only quoted in England[1] that the inexperienced might be cautioned against it.

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[1] It is hardly necessary here to indicate the details of this system, which comprehended strict rules for the conduct of each day. Between the daily bleedings 30 leeches were to be applied to the painful side. The slight cases would yield to three bleedings, the graver might require seven or eight or nine in addition to the leeching. If on the third day—contrary to the asserted rule—the disease still survived, general bleeding (of 3 or 4 palettes) was to be at once repeated. In the rare instance of still further resistance it is usually best to abandon blood-letting and apply a large blister.

[2] See Watson's 'Lectures on Medicine,' vol. ii. art. 'Pneumonia.'

Views as to blood-letting.

The doctrine of that day was in the main that of Laennec. It was precise and definite, and obtained a body of sanction hardly attainable upon any therapeutical question at the present time. Bleeding being the great remedy, the earlier it was practised the greater and the more certain the relief. Its amount was to be governed in each case by the impression produced upon the system, measured partly by the sensations and conduct of the patient, partly by the greater softness and fulness of the pulse, or, these signs not appearing, by the approach of syncope. The effect of a first bleeding in removing pain and dyspnœa was to be carefully watched. Usually a return of these symptoms would necessitate its repetition within four or five hours. Supplementing such treatment, cupping, or the application to the chest of a large number of leeches, was recommended. The diet was to be 'low and unstimulating.'

As the disease advanced, so, it was believed, did the abstraction of blood become less efficacious, but even with a solid lung bleeding was of use as 'tending to prevent the extension of the inflammatory process.' Yet a time might come when the shock of bleeding could no longer be borne, and other less powerful antiphlogistic remedies had to be substituted. Of these tartar emetic was appropriate for the stage preceding hepatisation, and mercury for hepatisation itself. The first, in frequent doses of a third of a grain (guarded at first by laudanum and increased, if necessary, up to two grains per hour), would often put an end to dyspnœa and allay any '*disposition to rekindle*' on the part of the inflammation; the second (blue pill or calomel, in small and repeated doses of a grain or two), was to be so administered as to render the gums tender as quickly as possible. Even should the lung remain solid under this treatment, it was still to be persevered with until the pulse and the face betokened the approach of death. Such was the teaching of the schools in London and Edinburgh thirty years ago.

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[1] 'I advert to his opinion,' said Sir Thomas Watson, lecturing at King's College in 1838, and alluding to M. Louis, 'merely to caution you against being misled by it, as you might otherwise be, considering his well-merited reputation as an exact and faithful observer.'

French teaching in 1841.

M. Grisolle in his work upon Pneumonia published in 1841 somewhat modified these views. Though favourable still to bloodletting, he for the first time fully recognised in distinct terms what Rasori had already expressed in words but denied in practice, namely the natural course which the disease was destined to fulfil, and which, whether grave or not, the conduct of the physician could not appreciably alter.

Bleeding and antimony variously employed.

Thus, although bleeding in some sort was during a long period the universal remedy for inflammation, and especially for lung inflammation, it must be noticed that it was not practised always to the same extent or to accomplish the same ends. As medicine advanced, the same weapon, the lancet, was put in succession to several uses. Bouillaud, by a strange figure, strangled pneumonia with it by repeated blows, Rasori, hardly less prodigal of blood, confessed himself unable to accomplish so much, while both Chomel and Grisolle clearly recognised that, whatever the method employed, pneumonia had its period of rise and decline notwithstanding. It is the same with tartar emetic. The one drug sufficed to give effect to many theories. Between the toleration and high doses of Rasori and the small diaphoretic doses of modern practice there is expressed a world of gradation or revolution in medical opinion as to the right treatment of the disease.

Yet while it remained the orthodox teaching up to 1847 (the date of Sir T. Watson's 3rd edition), that general bleeding was necessary in pneumonia, and that the quantity of

Substitution of tartar emetic for venesection.

blood taken should always be large, already there were signs of schism.[1] Louis had ventured to doubt the use of venesection except it could be practised early. Grisolle had convinced himself by the contemplation of Bouillaud that bloodletting did not effect all that was alleged of it. In the meantime attention had been directed to the virtues of tartar emetic given in large doses after the method of Rasori, and, as in the case of every drug brought prominently forward for a new use, assertions were now made in regard to antimony which we may well believe to be extravagant. Yet it was not unreasonable to hope that tartar emetic might at least aid, and in part spare, the work of the lancet. Already Laennec had announced that with moderate bleeding and tartarised antimony his success had been greater than with bleeding alone or any other method. Louis had recorded in detail the sudden relief that followed large doses of the same drug in critical cases which had been repeatedly bled without benefit, while Trousseau, subscribing to the same treatment, had met with but two deaths in 58 persons in 1831 and 1832.[2]

Bleeding relinquished from the absence of suitable cases.

Influenced mainly by such testimony, our own English practice drifted into a middle course, using bleeding a little and antimony a little, with a much diminished trust in the efficacy of both. And now, with the abandonment of heroic treatment, there concurred a much lowered mortality. Pneumonia ceased to be the formidable disease it had once been.

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[1] See Balfour, 'Edinburgh Medical Journal,' 1858, p. 214 *et seq.*, 'Hæmatophobia.'

[2] He admitted a less success by the same means in 1839, and believed that owing to the circumstances of the time bleeding became necessary. Yet of the effect of antimonials he still speaks (as indeed does Grisolle himself) in terms so high that he foresees that a future generation will tax him with exaggeration. Nothing is more remarkable than the rapidity of recovery. 'There is no convalescence. Three days suffice sometimes to bring back the patient from the gate of the grave to a state of health.'

The tendency to recover became more conspicuous than the tendency to die; the mortality, so to speak, became tolerable.

We cannot doubt (what, indeed, the literature of epidemics strikingly illustrates), that the mere death-rate of a disease will influence its treatment beyond what reason can justify. The remedy must be made to appear to the public eye proportionate to the malady. As the latter becomes less destructive a relaxed vigour in treatment is likely to ensue, which, though really the consequence, may easily be mistaken for the cause of the amendment. The publication, therefore, at this particular epoch of the disease, of certain statistics (as of Skoda in 1843–6, of the homœopathists in Vienna, of Barthez in Paris, and more recently of Dr. Hughes Bennett in Edinburgh), proving that the subjects of pneumonia might survive without medical interference, was taken to prove that the affection did best when left to itself.[1]

The progress of scepticism.

The history of this change in opinion is to be sought in periodical medical literature between 1847, when Dr. Balfour called attention to the treatment of Skoda,[2] and 1866, when Dr. Markham, in the Gulstonian Lectures before the College of Physicians, discussed the reality of the asserted change of type in disease. To the new views perhaps Skoda was the pioneer. Losing faith in active depletion he confined its employment to the period preceding hepatisation, and then used it only when the fever was high and dyspnœa urgent. In his treatment by corrosive sublimate (which he gave 'to reduce the plasticity of the blood'), he had a mortality of 1 in 7 with 392 patients, a result 'particularly favourable' at the time, especially as compared with the Edinburgh Infirmary (1839–1844) where the death-rate upon the old treatment was, as it seems, unprecedentedly high, namely 1 in 2·78.[1] Skoda, however, did not so much contend for a larger number of patients saved, as for the more speedy recovery of those that got well.

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[1] See Appendix G, 'Statistics of Treatment.'

[2] Balfour on the Practice of Skoda, 'Ed. Med. and Surg. Journal,' 1847, pp. 397, 398. Dr. Markham's Lectures are published in a pamphlet.

Asserted results of homœopathy.

The propriety of non-interference was further shown by the statistics published about the same time by Dr. Fleischman, showing a mortality of less than 6 per cent. obtained through homœopathy. Some doubt has been cast upon the accuracy of these figures, which provoked at the time much angry controversy. It would be profitless to discuss them now; right or wrong, they had no small share in directing attention to the possibility of abandoning active remedies for pneumonia, and thus helped to lay the foundation of the so-called expectant method.

Dietetic treatment.

Acting upon this hint, Dietl, in 1844–6, employed what he called a dietetic treatment in 189 cases, with a mortality of 7·4 per cent. He was less fortunate in a second series of 750 cases, where, nevertheless, the death-rate of 9·2 was more favourable than either Rasori's with high doses of antimony, or Bouillaud's with his repeated blows. There are no means now of analysing Dietl's results. As is usual, the treatment he pursued was less successful in the hands of his successors. It accomplished no further result than to encourage further trials. There was at least evidence enough to show that pneumonia might run its course without medicine, and that the result of non-interference, as compared with the most active depletion, was not necessarily disastrous.

Statistics of Barthez.

But the testimony did not end here; it was soon admitted to be the *habit* of pneumonia to recover. With patients from two years old up to fifteen it was asserted to be very rarely fatal. Out of 212 such cases Barthez reported that he lost but 2, this low mortality arising, not from any measures of his, but from the benignant nature of the disease.[2]

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[1] Bennett's 'Restorative Treatment of Pneumonia,' p. 41.

[2] See the statistical table, Appendix G.

Similar testimony was borne by England and Scotland. Never, under any system, homœopathic, allopathic, jugulant, or expectant, had such results been made public. Was this happy change due to an alteration in the nature of pneumonia itself, or in the current nomenclature, or to the fact that heretofore the methods supposed to be beneficial were in fact injurious?

The 'restorative' treatment.

To that question, difficult as it may seem, there were some who ventured to return a very positive answer. That pneumonia had never been so little fatal as in a certain period during which active medication had been withheld, was given in the facts. That to abstain from active medication, of whatever kind, was the best treatment for pneumonia was announced as the just conclusion. And soon, as though it were a corollary to that proposition, it was further declared that in this particular affection, while drugs were useless and worse, food was of exceptional benefit.[1]

Now it is not more reasonable, *à priori*, to assert that pneumonia may be conducted to a safe issue and have its mortality annulled by a uniform practice of feeding than it would be to maintain that its progress may be arrested by the uniform draining of blood. It is even less reasonable. For while blood may be abstracted without limit from every living body, the power to swallow food, and, much more, to retain and assimilate it, is wont to disappear very early in acute illness, and when the needs of the body are the sorest. The treatment is admirable for the favourable cases, but the very terms of its statement imply that its application will be limited to these. 'Good nutrition' and 'exalted vital force'

[1] Though it falls to my lot to question some of Dr. Hughes Bennett's propositions, I am not ignorant of the debt which medical science owes to his labours, and especially of the more enlightened treatment of pneumonia which has resulted from these views of his, which nevertheless, in some of their details, seem to me open to criticism.

are likely to be associated with such examples of pneumonia as recover; for the rest, the mere insertion of food into the mouth does not ensure nutrition or promote vital force. The possibility of feeding is past.

In speaking of the restorative method, therefore, it is necessary to distinguish between actual statistics and the arguments used to support them. The one must not be allowed to supplement the other. The plea that small numbers may suffice because the truth of the principle is self-evident must be rejected. The numbers must stand by themselves and the arguments by themselves.

Dr. H. Bennett's views and statistics.

Reading now the 129 adduced by Dr. Hughes Bennett in support of his method, but without his commentary,[1] this is what we find. The average age of the patients was 30½; 125 recovered and 4 died, a percentage (excepting Barthez with children), by far the smallest we have yet met with; 88 of the patients were under 40, 24 between 40 and 50, 13 exceeded 50, 2 of them being between 60 and 80. Of the 125 that recovered, 105 were simple and 20 'complicated.' Of the former, 79 were single and 26 double. The duration of the disease on the whole, on an average, was 14¼ days.

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[1] See Dr. Hughes Bennett on the 'Restorative Treatment of Pneumonia' (pp. 45–50.) 'This treatment,' says he, 'may be said to render the mortality in true cases of pneumonia *nil.*' 'The formation of pus is the normal and necessary transformation of the solid exudation, whereby it is broken up and caused to be absorbed.' Pus cells, it is explained, must be regarded as living growths, and 'as such require an excess of blood, good nutrition, and exalted vital force to hurry on their development and carry them successfully through the natural stages of their existence.'

The following extract gives Dr. Bennett's method in his own words :—'To further the necessary changes which the exudation must undergo I content myself with giving salines in small doses during the period of febrile excitement, with a view of diminishing the viscosity of the blood. At the commencement of the treatment I order as much beef-tea, milk, and other nutrients as can be taken, and, as soon as the pulse becomes soft, solid food and from 4 to 8 ounces of wine daily. As the period of crisis approaches I give a diuretic consisting of half a drachm of nitrous ether, and sometimes ten minims of colchicum wine three times daily to favour excretion of urates. . . . It differs entirely from the expectant treatment in the care which is taken to nourish the weakened frame from the beginning,' &c., &c., *loc. cit.* p. 51. It is added moreover, that by this treatment (1) the blood is rendered less viscous by salines; (2) the system is fed by means of wine; (3) urates are excreted by means of colchicum and nitrous ether.

These numbers (given by Dr. Bennett with much detail) are sufficient to show beyond doubt that, in respect both of duration and mortality, the successful issue of these cases is exceptional as compared with other statistics.

Analysis of his tables.

It is to be regretted, therefore, that results so satisfactory cannot in strictness be adduced to support any treatment in particular unless it be the giving of alcohol. These tables do not even illustrate, for good or bad, the effect of any single treatment, 'restorative' or otherwise. Thus, taking the twenty worst cases, or, at least, those that appear to be the worst, upon the evidence of pulse, respiration, and extent of lung engaged,[1] it is only in 7 that the 'nutrient' system was applied. The other 13 were treated with bleeding, or antimony, or laxatives, or salines. The worst cases got alcohol, some of them in considerable quantity. One of these is stated to have been 'saved' by half-ounce doses of wine every half-hour. If the tables had been framed upon Grisolle's plan of excluding those that would almost certainly recover anyhow, it would have been reduced to 53;[2] if it had excluded those who were not brought under Dr. Bennett's treatment till after six days' illness it would have been reduced to 76; if it had included those only upon whom the nutrient system was in fact fully tried along with wine, but with no active drugging, it would have comprised but 46. Of the treatment by nutrients apart from wine there is hardly a case, certainly not a grave case. What the table shows chiefly is the treatment by alcohol, in moderate quantity for the most part, but indefinitely increased when occasion seems to require it. The drug which, after alcohol, is most conspicuous is antimony. Such statistics illustrate only what was said at the beginning, that the course of treatment, inasmuch as it follows the course of disease, is seldom, in fact, uniform.

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[1] See Appendix G. for an analysis of these.

[2] Only 53 had on admission a pulse over 100.

Dr. Waters' cases.

Similar objections would apply to the tabulation of this disease by other physicians, as, for instance, by Dr. Waters of Liverpool;[1] yet this author, as I gather, does not publish his table to illustrate the success of a particular treatment, but rather to show the usual course and small mortality of the disease. The cases are too few and are seen too late to serve for more than this. The remedies used are simply adapted to the particular wants of the individual. Beef-tea is administered as such, as the common food of the sick; salines are used in obedience to a common routine, apart from the 'viscosity of the blood,' and, in the important matter of direct stimulation, alcohol is given or withheld as the circumstances seem to require; in the last resort, it is trusted in entirely as the sole stay and hope of the patient.

Thus the question as to the actual numbers which might fairly be regarded as sufficient to illustrate the operation of a particular course of treatment does not in fact arise, since in such numbers as we get, no particular course of treatment is actually put in practice.

Statistical evidence for drugs.

Let us turn to the evidence for or against the use of special drugs. It would seem more feasible to study the conduct towards a particular disease of some active agent, given to one group of patients and withheld from another, rather than to judge of the benefit, more or less, of a description of nutriment like beef-tea, given in the present day to all alike in such measure as they can receive it. Nevertheless, as a matter of fact, there is nothing on record in reference to such agents (with the exception, indeed, of antimony) that need detain us for a moment. The salts of iron and of copper, acetate of lead, veratria, alkalies, chloroform inhalation, and other things have, indeed, been hypothetically recommended, and statistical evidence has been adduced in favour of some of them. Thus Zissel in 1848–50 with acetate of iron and acetate of copper lost 4 cases out of 93, or 4·4 per cent., 70 of these being under 20 years old. Sauer with sulphate of copper lost 3 out of 56, or 5½ per cent. Zeigelé in 1848–9 with hydropathy and young patients lost none out of 40, or nothing per cent. It is idle to accumulate figures like these, nor is it too much to say that there is no published account respecting any of these methods entitled to consideration.

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[1] See Appendix G.

Drug remedies used in virtue of their physiological action.

It may be urged, however, that there are other modes of estimating the value of drugs besides the numerical, that, for example, a particular medicine may be rationally selected in pneumonia on the ground of its ascertained physiological action upon the organism, even though statistical results in reference to the particular disease are not obtainable. In this view digitalis, aconite, belladonna, quinia, veratrum, have been employed in pneumonia in virtue of some influence they are believed to possess over function, whether in moderating the pulse, or reducing the body temperature, or producing diaphoresis. It would be out of place to enquire into the validity of this application; it will be enough to enumerate some of the medicines which have been thus used, leaving the rest to the judgment of the reader.

Digitalis.

Digitalis had already been largely tried by Wunderlich, Ziemssen and others, to reduce the pyrexia of typhoid. Bleuler making trial of this same drug in pneumonia got a mortality of 21 per cent., as against 14·5 per cent., the mortality without it. He did not himself note any effect of the medicine in cutting short the inflammation, but remarks upon the early period of defervescence in his own cases, viz. from the fifth to the seventh day. Bleuler gave as much as half a drachm of digitalis daily, and elicited in some instances the poisonous effects of the drug. M. Piory-Sancerotti gave foxglove in 35 cases of pneumonia, and concluded thence that as an antiphlogistic 'it was less rapid but more durable than leeches.' Furthermore, the use of digitalis in pneumonia has the approval of Niemeyer, an approval which Stillé insists that there is no direct evidence to justify.

Veratrum.

Veratrum viride and veratrine have been recommended in pneumonia to reduce pyrexia. Dr. Kiemann gave the former in 40 cases, 5 of whom (or 12·5 per cent.) died. The action of the drug as an anti-pyretic was marked and invariable. There is other testimony by Drasche and Vulment to the same effect, though the former adds that veratrum rathers postpones the process of resolution.[1]

Veratria.

Veratria, on the other hand, upon the evidence of Kocher,[2] shortens the duration of this process. It reduces the pulse and temperature, and sometimes appears to cut short the disease and prevent consolidation. This author alludes, however, to the dangerous depression produced sometimes by veratrine, and believes that the drug cannot be safely given in larger quantity than one-twentieth of a grain at intervals of from one to two hours, until an obvious impression is made on the pulse or the temperature. The good results obtainable from veratrine may be promoted, according to the same authority, in severe cases by bleeding. Other authors have made use of the drug and generally commend it, as Aran, and Vogt, and Trousseau. Its power to reduce temporarily the frequency of the pulse is pretty well established, but it is agreed that its effects are not constant, that it has to be used with great caution, and is apt to cause diarrhœa and vomiting.

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[1] Phillips, 'Materia Medica,' art. 'Veratrum.'

[2] Quoted by Wilson Fox, p. 699, Reynold's' 'System,' vol. iii.

Aconite.

Aconite has been much extolled of late as a reducer of body temperature. As regards its use in pneumonia Dr. Phillips[1] records that in 9 cases under his own observation the temperature fell in from 3 to 6 days and the pulse with it. From 11 other cases he judges that aconite has no effect in removing consolidation, but controls and removes the 'tendency to spreading of the congestion.' Dr. Wilson Fox has given aconite in one or two cases, but could not observe any effect produced by it on the temperature. Dr. Anstie used to regard aconite as the most certain and reliable of diaphoretics.

Belladonna.

Belladonna has been recommended in pneumonia, I know not on what precise grounds.

Cold.

In the sense of a cure for pneumonia the direct application of cold, as commended by Niemeyer, requires to be mentioned along with the drugs. The method consists in applying compresses to the chest wrung out of cold water and changed every few minutes. In a very large number of cases, we are told, the disease so treated terminates on the third day.[2]

Alkalies.

Alkalies have been commended in pneumonia. By the use of bicarbonate of potash in doses of from 5 to 30 grains, largely diluted, Dr. John Popham[3] believed that a sedative effect was produced, and that the 'physical signs became resolved.' As an instructive commentary upon this statement, and upon the assertion sometimes made as to the power of alkalies to prevent hepatisation, I would refer the reader to a case already recorded on p. 72.

Lastly, there is an enquiry, not by one individual but by

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[1] *Loc. cit.* p. 7.

[2] See Reynolds, vol. iii. p. 700.

[3] Brit. Med. Journ., Dec. 28, 1867.

Action of quinia upon pneumonia.

a Committee,[1] into the action of quinine in the pyrexia of various diseases. As regards pneumonia only one instance appears. Ten grains of quinia were given on the morning of the fourth day during the pyrexial remission. A reduction of temperature followed amounting to 2° of Fahrenheit. The 'effect' lasted 24 hours, the pulse and respiration being but slightly reduced. After 30 hours the temperature 'returned to its ante-quinine range.' In several other diseases though the pulse fell with the quinine, the respiration was quickened. This Committee adds, as respects quinia in its action in reducing temperature, what one cannot but regard as a significant statement equally applicable to the other drugs: 'There was no conclusive evidence that the quinia favourably influenced the duration or course of any of the diseases in which it was administered.'

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[1] See Report of Committee, 'Clinical Soc. Transactions,' 1870.

CHAPTER XV.

THE TREATMENT OF PNEUMONIA.

Circumstances of the patient—Indications for treatment—Of diet—The use of alcohol—Treatment of pleuritic 'stitch'—of sleeplessness—diarrhœa—prostration—Extreme dyspnœa—Question of bleeding—Active delirium—Application of ice to the head—The use of narcotics—Choice of stimulants—Stimulation by alcohol—Treatment directed against the pyrexia—The external application of cold—Waiting and watching—Convalescence—The sum of the treatment.

Circumstances of the patient.

Subject to such sources of error as have just been alluded to, and not unmindful of the force of prejudice, I venture to indicate that treatment of pneumonia which in the present circumstances I believe to be necessary and sufficient. There are, to begin with, certain well-established facts to be kept steadily in view. Such are our present knowledge that the natural course of the disease is commonly not destructive; that in childhood, beyond infancy, its mortality is very small, while at the latter end of life it is very large; that there are particular circumstances unfavourable to recovery, such as alcoholic intemperance, and destitution, and particular antecedent states, such as acute rheumatism, which may render it rapidly fatal. An ordinary case of pneumonia it is difficult with ordinary care to lose; a case under these exceptional conditions it is difficult with the best exercise of care, I do not say to save, but intelligently to attempt to save. In all the fluctuations of statistics may be discovered examples of pneumonia of the purulent type, now prevalent and now rare, invariably, probably inevitably, fatal. Until these can be got rid of, or until they can be discriminated and placed by themselves, it is improbable that the mortality will ever be reduced, as Dr. Hughes Bennett hopes, to nothing.

Treatment indications.

To the domain of treatment, however, it legitimately belongs to place the patient under the best conditions[1] for enduring an illness whose probable course and special dangers and duration are approximately known, to relieve pain, to secure sleep, to feed, and lastly to have recourse to exceptional measures for relief when life is immediately threatened by dyspnœa. The treatment of pneumonia is to be governed by the patient's general condition and not by the physical signs obtained from the chest. Measures directed to the lungs are irrational and often mischievous. First in importance in pneumonia, as in continued fever, is the question of diet.

Dieting.

For it happens not seldom that total want of appetite, such aversion to food[2] and real inability to swallow it as renders the ‘restorative’ treatment of limited application, is a frequent source of trouble. I think the use of drugs in these circumstances should be almost restricted to those that have good repute either as stomach tonics, such as hydrochloric or nitro-hydrochloric acid with gentian, or (if these are indicated), as antacids or carminatives, such as the effervescent saline draught, the object being to obtain from medicine what medicine is in fact able to afford, an ‘adjuvant’ to the digestive process.

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[1] I deem it unnecessary with the instructed reader to lay stress upon the importance of pure, *i.e.* often renewed air, and carefully regulated temperature. I have often thought that in these days of rapid and easy transit the main 'indications' for treatment of pneumonia in our large towns might be best met (weather and other circumstances permitting) by carrying the patient at the very outset of his disorder into the pure air of the country. Besides removal from what is directly harmful, noise and vitiated atmosphere, it can hardly be that the prompt benefit which most of us experience upon leaving town when in ordinary health or suffering from chronic disease, would be wholly unfelt when acutely ill.

[2] It is important to learn how the patient is apt to be affected in this respect by particular drugs, and moreover to feed him—so far as is consistent with a nutritious fluid diet—as he is wont to be fed, that is, according to his known tastes in health. When the sick man turns from what is offered him and exhibits real distress at the approach of nourishment, it is forgotten that to the sound man port wine in the

Early appearance of liver symptoms.

We see occasionally at the beginning of an ordinary pneumonia a loaded tongue, headache, constipation, bilious vomiting or nausea, yellowness of the conjunctiva, sometimes even jaundice, in a word, all the array of what is commonly known as a ‘bilious attack’ and which, appearing in this combination, is chiefly troublesome as rendering the patient averse to food or unable to retain it. I think these symptoms are to be treated here in the same manner as where they occur alone, namely by giving three or four grains of blue pill with six of colocynth, or the blue pill at night and the senna draught in the morning. An operation of the bowels is almost always salutary in these circumstances, and the blue pill, I must still believe, has a special virtue.

Use of alcohol.

With the object of promoting or creating appetite it is sometimes desirable to give some form of alcohol in moderate and measured quantity, as dry sherry or light burgundy. This is especially the case when the patient is habituated to such kind of stimulation. It is seldom, however, at this period of the illness[1] that wine is really

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early morning, or the swallowing of beef-tea immediately on awakening, would hardly be grateful.

[1] See the experiments by Dr. Parkes with alcohol administered to a healthy man (‘Proc. Royal Soc.,’ May 19, 1870). ‘Two fluid ounces of absolute alcohol seemed to increase the appetite, with larger quantities it lessened or destroyed it. The commencement of narcotism marks the time when appetite and circulation begin to be damaged.’ What

necessary as a direct stimulant in respect of its alcohol. Of this use I must speak presently.

With these means of husbanding the strength and supplying the extraordinary demands of the system, must be conjoined every appliance that may promote rest. It can hardly be doubted that the effort of speaking is injurious. The questions addressed to the patient should be few; he should be cautiously and dexterously moved for necessary auscultation, and kept for as short a time as possible in a constrained posture unfavourable for the respiratory movements.[1]

Treatment of 'stitch.'

There are certain symptoms proper to the disease which are chiefly harmful in their power of depriving the patient of necessary sleep. One is pleuritic stitch; another the constant harass of cough. Remedies are not altogether powerless against either of these, yet sometimes they remain unrelieved because unnoticed. For in pneumonia, as we have seen, there creeps over the sufferer a certain lethargy, so that his personal complaint ceases to be a nice index of his actual suffering. This catching pain with inspiration is often obviously lessened by the application of moist heat.

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occurred to this man may not be true for the whole race, and what is true for health may cease to be true for disease. It is unnecessary, I believe, to make wine a part of the ordinary treatment of pneumonia, and impossible as yet to lay down exact rules in regard to its use. In, small quantity (as for instance, from two to four ounces of dry sherry or of champagne) it can seldom, unless very distasteful to the patient, do harm. And for much of its effect—as for this one upon the appetite—we must have recourse to common observation rather than scientific rules. Alcohol long continued is eminently injurious to children. On the other hand it cannot be suddenly withheld from those who have habituated themselves to the over-use of it without producing a depression which, even for its mental part, we must desire to avoid in a disease like pneumonia.

[1] It will be prudent sometimes (although the plan is never satisfactory to the physician), to examine the chest without raising the patient.

The swathing of the chest in an ample fold of flannel from which water quite hot has just been wrung, with diligent renewal at equal intervals, so as to maintain for a full hour a continuous application of heat at a uniform and grateful temperature, is almost always serviceable. Yet a nurse's notion of a hot fomentation is often quite different from this. To accomplish it with the least disturbance of the patient and without leaving the bedclothes wet needs practice and address. Unless done deliberately and with intelligence it is useless or worse. Sometimes it so happens that a dry heat, as the application of flannel from the oven, or of a hot-water bag, is of more use than fomentation. Both are valuable, and the more to be insisted on because with some minds their simplicity is against them. The manner of performing these small offices, the skill and dexterity which can perform them efficiently, with no disturbance to the patient from what is being done, but only a sense on his part of benefit received without his co-operation, comprehends no small part of the total amount of service we can render to those who are undergoing pneumonia.

But the relief afforded by these appliances is apt to disappear so soon as they are removed. If, therefore, pleuritic pain with inspiration form a prominent part of the patient's distress (which, though not the rule of the disease, is not uncommon), fomentation may be supplemented by leeching. That the abstraction of blood from the neighbourhood of the pain should give more relief than its withdrawal from a vein in the arm, is a fact not easily understood, but generally acknowledged. It is indeed the one feature of treatment which has lived through all the changes of practice, and still survives unsupported by any theory, with no better justification than its obvious good service. Some special advantage may perhaps be gained by thus adding a new sensation at the very spot of the existing one. The leeches, three or four, should be applied as near to the pain as is consistent with their being also on the anterior half of the chest, so that during their application the patient may lie as best suits him, and afterwards not rub the place of the bites in moving. After the leeching hot fomentation may be re-applied, and at this particular juncture, if by reason of pain sleep has long been absent, an opiate draught of moderate strength (opium or morphia for greater certainty rather than chloral), operating with the soothing warmth and concurring with diminished suffering, may procure some hours of tranquil sleep, which before a larger dose might have failed to obtain.

Sleeplessness.

For in this matter of sleep it is far better to obtain it indirectly by removing or mitigating the pain that prevents it than by dulling the senses with narcotics. Absolute ease or sound refreshing sleep we are hardly to expect at this stage. To seek to abolish pain by means of large doses of opium or chloral or by hypodermic injection is to prefer present comfort to future safety. That amount of narcotism which, in spite of pleurisy and irritable cough, will so far blunt the senses as to admit of prolonged sleep can never be induced without mischief. Such sleep is unrefreshing, and the waking from it is often the waking to an aggravated suffering which has thus been allowed to accumulate unobserved. At the same time the early resort to narcotics, from the habit that it genders, is an evil provision for the impending needs of the patient.

Both in soliciting sleep and in attempting to arouse the appetite the force of use and custom must be regarded. The recurrence through years of bed-time and meal-time at fixed periods of the twenty-four hours renders these the times when the body is naturally disposed, or least indisposed, either to sleep or to eat.

Cough.

As with sleep, so with cough, it is quite possible to be over-busy about it. Except with children, where it sometimes becomes paroxysmal, this symptom is seldom a source of great distress. When it is so it is the custom to add to the medicine some reputed expectorant, or to give separately some form of linctus. I have not found that much benefit follows these measures; at any rate, such supplementary drugging is not to be allowed to supersede or interfere with the cardinal objects of treatment. I prefer in the case supposed to try the effect of breathing for a while moistened air, driven into the mouth by means of the 'steam-draught inhaler,' which may be impregnated with the succus conii, though it is true that the proceeding sometimes wholly fails in its purpose.

Diarrhœa.

The pneumonic fever, like other pyrexia, is usually accompanied by constipation; sometimes, however, the early days of the disease and sometimes its crisis are marked by the presence of diarrhœa. This may arise from purely accidental causes, the alteration of diet and mode of life, or the too frequent administration of nourishment. Sometimes it is the consequence of domestic medicine taken at the outset to work off the first feeling of illness, and which, from a prevailing instinct, is almost always aperient. It is reasonable to suppose that in these ways statistics relating to this symptom rather overstate its natural frequency. Grisolle observed it in 9 out of 51 cases. It is said to be more common with children than with adults, although probably the statement would not hold in reference to true pneumonia. Diarrhœa at this period is rather a source of discomfort than of danger, and seldom needs the employment of active astringents. A careful restriction of the diet, warmth to the abdomen, the giving of food with observation, at such times in the day as the bowels are found to be least irritable; a restricted use of such fluid as is not food, with a tablespoonful of the aromatic mixture of chalk after each liquid motion, will commonly suffice. If the diarrhœa persist, laudanum may be given in some vegetable bitter in doses which may be astringent but not narcotic. For the short time such medicine is necessary it is desirable in an ordinary case to suspend other drugs and avoid the risk of wearying the patient with over-service. Diarrhœa continuing without abatement through the first week will lead to the suspicion of commencing ulceration in the small intestine, and this, in its turn, to the probability of tubercle being present in the lung, or of the supposed pneumonia being more properly a process of alveolar catarrh with which diarrhœa is not uncommon. In the latter stages of fatal pneumonia, however, diarrhœa sometimes appears with the closing symptoms and lasts till death, where there is no intestinal lesion to account for it.

With no further assistance from drugs than has just been detailed the large majority of persons attacked with pneumonia (old age excepted) make a good recovery. In from three to eight days the fever slackens or disappears, and the convalescence which shortly follows proceeds commonly without interruption, and without obvious help from medicine, until it ends in health.

Modes of danger.

Yet it is not always so, nor is it possible at the outset to discriminate the cases that will do well from those that will do ill. Untoward symptoms may declare themselves suddenly in asphyxia or syncope, or the fever may be unusually prolonged so as to threaten death by exhaustion. Sometimes, indeed, it is obvious from the first that the attack is severe and its issue doubtful. This may appear either from the local or the general side of the affection. The lung implication may be extensive or it may involve both sides, or be conjoined with pericarditis, or, commencing at one spot, it may rapidly spread in the manner described by Trousseau.

On the other hand, apart from the physical signs, there may be that in the aspect of the patient which bodes ill.

Of bad augury always is that condition which assimilates some cases of pneumonia so closely to continued fever; listlessness and indifference, with blunted perception and occasional mental wandering.[1] Whether the local mischief be extensive or not such patients often die, these symptoms appearing to accompany rapid breaking up of the lung. If with such a condition the patient is known to have been of drunken habits, or still more, if with habitual excess in alcohol there has concurred habitual deficiency of nitrogenous food, the speedy spoiling of the lung by purulent conversion and consequent death may be regarded as probable. If we could exclude cases like these the mortality of pneumonia might be very materially reduced. Unfortunately we can neither exclude them nor distribute them evenly. They occur very irregularly, sometimes in batches and sometimes widely separated, and thus contribute to produce that inequality in the death-rate of the disease at different times and places which has been so often remarked upon and so commonly attributed to some drug influence.

Extreme prostration.

That the power of medicine, or perseverance in administering food or wine, can have much influence in altering the course of such a pneumonia I do not personally believe. Yet inasmuch as instances of the kind do not at once declare themselves, there can be no justification while life lasts for regarding *any* case of pneumonia as absolutely hopeless. Our duty is to seek to relieve each symptom as it arises, however urgent or even desperate may be the general condition.

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[1] It has been noticed already that these signs are oftenest observed with pneumonia at the apex, and so far it may be true that this form of the affection is exceptionally grave. Yet pneumonia of the apex, as such, is not more formidable than of the base. The cases which are placed in this category are often discovered post mortem to be instances of *spreading* pneumonia also, the affection not being recognised until the apex is reached. On inspection, the whole of one lung is found to be involved, the disease being most advanced, not at the apex, but at the upper part of the lower or lowest lobe, which thus appears to have been its starting-place. See p. 66.

Dyspnœa.

As for dyspnœa, its importance as a symptom must be estimated not merely from the character or frequency of the respirations, but as well from the whole behaviour of the patient in respect of the respiratory function. Sharp pleuritic pain may render the breathing shallow, and catching, and frequent. No inspiratory act can be completed owing to the pain it gives, and the patient seeks to make up by pace for what is wanting in completeness. Such a method of breathing, registered according to mere frequency and compared with the rate of the pulse, would certainly mislead. Dyspnœa brought about in this way, dyspnœa that is *felt*, that produces, indeed, a lively sense of distress, is better than dyspnœa of the same or even a lower rate which is not actively distressing to the patient, although it is obvious to the sight that it is not more efficient than the other.

It is upon such considerations that the treatment of this symptom should be based. If within the first forty-eight hours considerable dyspnœa[1] (let us say between forty and fifty respirations per minute) correspond with a large extent of lung mischief, and if the stethoscope indicate that consolidation is only commencing, while at the same time the patient is attentive to the sensation breathlessness, yet able to take nourishment and to get snatches of sleep, then, I believe, looking to the usual issue of such cases, that it is best to adopt no more than the palliative measures already indicated, waiting for the change that may be expected within a fixed period. If, however, the patient be not encountered till later, say on the third or fourth day, a large area of his lung being now solid, with the dyspnœa he has had from the beginning still extreme, I believe then that it is wise and prudent to adopt the only known means of relieving such dyspnœa, namely bleeding. The immediate (although too often temporary) benefit got from timely blood-letting is enough, in my judgment, to outweigh any consideration of the possible harm to be done by it.

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[1] It will be remembered of course that, *cæteris paribus*, the respiration of children will be more disturbed than that of adults ; that causes independent of the pneumonia, such as pregnancy, deformity of the thorax, dilatation of the heart, anæmia, and so forth, may any of them contribute to heighten the breathlessness. In may be mentioned moreover that the *nervous* element is never to be overlooked in connection with this symptom. It is said that hysteria disappears before acute disease ; nevertheless hysterical women and 'nervous' persons of both sexes may have extreme dyspnœa, as I have seen, with only slight pneumonia. On the whole the respiration is to be judged of by what it *does* in fulfilling the wants of the system as regards aëration rather than by the sensations aroused by the act itself. Dyspnœa is a symptom difficult of analysis, as including both act and sensation.

Question of bleeding.

It is difficult by words in a book to make one's meaning clear upon such a point, or to discuss it in its several bearings. What *is* urgent dyspnœa, and what is the precise period at which it begins to be dangerous? I would put it (I mean my own opinion) in this way. When dyspnœa is the prominent symptom, so that from its presence mainly the patient is denied rest, and when the physical signs indicate that the affected lung is solid, or mostly solid, then, in the knowledge that relief is almost always brought in that way, and in the belief that it is in any case harmless, I would bleed from the arm to eight or ten ounces. Nor would I hesitate to repeat such bleeding where the benefit of the first operation had been marked at the time, yet after a while dyspnœa had again become pressing. Here is no theory of reducing inflammation, no promise of arresting or strangling the disease, or even of relieving the right side of the heart; only a procedure for the gaining of time where a certain period has to be lived through, a measure of relief which, imperfect and brief as it is, may yet serve its turn.

But while the call for aid which is prompted by the patient's active distress from dyspnœa is almost always satisfied for a while by means of the lancet, it is otherwise where the very same need of air exists in one who is inattentive to his own suffering. Little can be said, I think, either for bleeding or any other procedure when extreme dyspnœa goes along with extreme prostration, and the patient is at once breathless and unconscious.

Treatment of delirium.

Leaving that for the present, there are other phenomena of pneumonia which, by their prominence or persistence, may claim attention. One of these is *delirium.* Excluding cases of alcoholic excess, active and persistent delirium is rare in the disease, but most common when the apex of the lung is affected. I have already had occasion to refer to instances in which delirium appeared amongst the earliest symptoms.[1] Such cases, with others that might be quoted, prove that the symptom does not always indicate any special danger. Yet active delirium, inasmuch as it is here exceptional, should always suggest a careful scrutiny of the grounds upon which the diagnosis has been based. Typhus and typhoid fever with pulmonary congestion are frequently mistaken for pneumonia, and with these, as we know, especially the former, active delirium is quite common. Assured, however, that the disease is pneumonia, active delirium would next suggest alcoholic poisoning, or, failing that, some affection of the kidneys, the prominence of the symptom being then due to uræmic poisoning.[2]

Yet although delirium, beyond night-wandering, is uncommon, it may occur when we least expect it, and when there is no doubt that the pneumonia is simple and uncombined. Delirium, like pain, is more acute and more easily excited in one subject than another; with some it is aroused upon every accession of pyrexia.

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[1] See p. 67; also Appendix C, p. 262.

[2] It was so in my experience in a rapidly fatal case of pneumonia, marked by remarkably fierce delirium, in a man of middle age with granular degeneration of the kidneys.

Importance of feeding.

In dealing with watchful delirium everywhere it should be the first care of the physician to see enforced certain special directions as to the nursing of the patient:—absolute quiet, dim and even light, timely administration of food, and the constant presence of a face and voice familiar to the patient, and instructed by experience when to speak and when to be silent. Let such an one be informed that the symptom in question is more likely to yield from soothing and feeding[1] than from formal treatment. As for the latter it will often be found useful, especially where along with delirium there is headache, to apply a bladder of ice to the head and forehead, with the precaution, however, that the application is not to be continued indefinitely. Whether this measure give ease or not it should in either case be removed after a short application. To keep the patient for an indefinite period with his head supporting a weight of ice is likely to add to his discomfort, though he may want the energy or the sense to resent it.

Use of narcotics.

When this watching is found to be unaffected by these measures, and when the patient is not merely restless but actively delirious, then, whether or no the history can connect the attack with intemperance, it is proper, I believe, to give opium or morphia either by subcutaneous injection or by the mouth in *one full dose* (according to age), to be repeated, if necessary, after an interval of three or four hours. Chloral is too uncertain to be trusted at so critical a juncture, while the full dose of some form of opium is a better practice than that of repeated smaller ones, for these are apt to be the reverse of narcotising.

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[1] There is an admirable colloquial expression, 'too weak to sleep,' admirable not only for its truth but because it suggests the best of all remedies for such sleeplessness—food.

Narcotics in combination.

Delirium occurring early in pneumonia and while the lung changes are yet in progress, might suggest the use of tartar emetic and opium in combination, after the plan advocated by Dr. Graves [1] and more recently by Dr. Murchison, for the watchful delirium of typhus. I have small experience of this combination. It is alleged that the addition of antimony promotes the action of the opium, so that a smaller dose of the latter suffices. What is wanted in the condition we are considering is to make an impression by a single dose, yet to be as sparing as possible of the narcotic. Laudanum or solution of acetate of morphia in conjunction with a stimulant like spirit of chloroform, or sulphuric ether, or sal volatile, is a very serviceable combination in the circumstances of great prostration and total sleeplessness. If sleep should be secured through such means, yet the general restlessness continue, it will be well now to give for a while nightly doses of chloral. In the use and selection of such remedies it is always of advantage to learn how the individual in question is apt to be affected by narcotics. A knowledge of the action of these drugs *generally* requires to be supplemented by a knowledge of the nature of the recipient.[2] Otherwise the opportunity may be spent in vain experiment.

Never should direct narcotics be given in pneumonia without clear necessity, and the conviction, at the time, of their being in themselves evil. Yet when, upon such need as we are considering, they are had recourse to, they must be used in efficient doses. The good is only to be got by encountering the evil. Frequent repetition of minute doses of opium misses the desired end and is emphatically weak practice.

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[1] Graves's 'Clinical Lectures,' i. p. 257.

[2] I abstain throughout from writing prescriptions or naming doses. These must always have reference to the individual: we can but discuss general principles.

In the pneumonia of drunkards, 'complicated' as it is phrased with delirium tremens (where, that is, the delirium takes the busy form of that disease and the history of the patient points to alcoholic intemperance), a similar treatment is called for, but with a more restricted use of opium than with delirium tremens alone. The insidious character of this form of pneumonia has already been adverted to.

Narcotics inapplicable.

If, however, there be great prostration with the delirium, if, along with occasional accessions of strength which the patient uses to escape from bed, or, under some delusion, to resist restraint, there are all the indications of ebbing life,—a blue face, panting breath, sweat-covered skin, and soft, compressible pulse, then I think opium and all such things whose effect it is to deaden the senses to external impressions, must be peremptorily forbidden. The maintenance of life is now inconsistent with drugged sleep.

Means of stimulation.

Such cases are to be treated on the same principle as other instances of failing vital power, be the cause what it may. We strive, that is, to maintain life by stimulants, amongst which alcohol holds the chief place,[1] and for the rest we administer, upon a generally received tradition that rests upon no basis of exact observation, frequent doses of cinchona bark and ammonia. Sometimes it becomes evident *post mortem* that during the latter days of the patient's illness his condition was in fact past hope; that while we were pouring in brandy and bark his lung was already destroyed and his life forfeited. Sometimes, on the contrary, this extremity of danger is finally surmounted, just as it is, more often, in fever when death seems already in sight. It is not a hopeless condition. It has been recovered from now and again under every mode of treatment.

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[1] 'I have in one or two instances,' says Dr. Wilson Fox (p. 705, *loc. cit.*) 'given 36 ounces of brandy daily for several days consecutively in cases of pneumonia in drunkards, every attempt to diminish the dose being immediately marked by dangerously increasing signs of asthenia; and it was only when the more marked evidences of prostration diminished that any symptoms of alcoholic intoxication were observable.'

In such circumstances the young practitioner is apt to be perplexed and wavering in the choice between a large number of agents, all of which are highly extolled. He flies from one to the other, and is disappointed with all in turn, for each is tried incompletely with only a half confidence, while the thoughts are employed with the next. Sometimes he forsakes them all in despair, and trusts solely to brandy; but more often the patient's death leaves him in regret that he made use of so few and never reached the right one.

Alcohol.

Musk, camphor, phosphorus, the cold affusion, stimulant applications to the surface with mustard and cantharides; the literature of typhus abounds with such remedies.[1] It is difficult to choose between them, each has its hour of popularity and its special advocates. For myself I will confess that I know of no stimulant in the fever-like prostration of pneumonia that seems to me so good as brandy; yet while I can recall cases that appear to have been kept alive by its use, I could never discover *how* alcohol becomes thus serviceable or what particular symptom was altered by it.[2]

But the disease may threaten death by the mere intensity of its pyrexia. Beyond the limits of ordinary fever there is

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[1] See Murchison on 'Fever,' *loc. cit.* p. 278. The terms in which such agents are praised are similar to the following, which refers to oil of valerian. 'The change following it was so unforeseen,' writes Bourralier, 'so astounding, that often I have heard persons who attended my visits pronounce the word resurrection.'

[2] Dr. Graves used to teach, quoting Dr. Armstrong, 'that alcohol does harm if under its use the tongue become dry and baked, the pulse quicker, the skin hot and parched, and the breathing more hurried, conclusions we can have no hesitation in accepting. It is now asserted (though not as yet proved) that the usual effect of alcohol in acute disease is to lessen the temperature and the urea discharge. The former of these effects is certainly accomplished sometimes, not always.

Treatment of pyrexia by cold.

it seems a region which men cannot reach and live. When the continued rise of the thermometer, therefore, seems to indicate that this fatal degree of heat is being approached, the plan has been proposed and adopted of cooling the body from without by means of ice-cold water. Upon the hypothesis there is pyrexia and hyperpyrexia. Different diseases reach different temperatures, but there is a temperature fatal to all alike, and where this is threatened we have

by quinine, which nevertheless is not otherwise of marked service either in pneumonia or continued fever. (See Committee's Report on Quinia, 'Clinical Soc. Trans.' 1870, and p. 220.)

My friend and then colleague, the late Dr. Anstie (who at the time of his death was occupied with researches upon the subject) published last year in a clinical lecture ('Lancet,' March 21, 1874) the particulars of a case of double pneumonia admitted into the Westminster Hospital, where 'with the area of inflammation extending,' the temperature fell continuously from the first day.' The patient (a fairly robust man of 31) was treated with six ounces of brandy daily. The urea discharge was markedly reduced at the same time. The man's habits in regard to alcohol are not mentioned. The author refers to a second case in which pneumonia coming on after several days of pleurisy and left to run its course up to the sixth day, was then given three ounces of absolute alcohol. The temperature 'continued almost undiminished for several days ; the urea fell on the first day of the stimulation from 1,348 grains to 1,317, and the succeeding days to 552·1, 467·2, 189·15. Dr. Anstie adds his intention of publishing a large quantity of evidence by which the general proposition is supported that 'in any acute pyrexial disease in which high temperature is conjoined with a large urea discharge, that conjunction is the strongest *primâ facie* reason for the administration of alcohol, and that a reduction of heat and urea discharge is to be regarded as evidence that the stimulation has been rightly adopted.'

With such observations must be joined those of Dr. Parkes * (already alluded to) upon the effects of alcohol in health, its not raising or depressing temperature, nor 'altering in any material respect the elimination of nitrogen from the healthy body.' Though here again it is obvious that the effects produced upon health do not necessarily indicate what alcohol might accomplish in disease to control undue urea discharge or reduce elevated temperature.

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* 'Proceedings Royal Society,' May 19, 1870.

resort to the only known means of reducing animal heat. To speak more particularly, while pyrexia[1] in pneumonia and acute rheumatism may be seen and tolerated at 104°, yet from this point to 109° is a temperature apt to be fatal, to be called hyperpyrexia and met by direct treatment. Now it happens in fatal cases of pneumonia with exceptionally high temperature, that death reveals a condition of the lung absolutely irrecoverable; the high temperature is but the index of a condition which must under any circumstances have ended in death. For myself I do not know of any case of pneumonia dying with such exceptional temperature that could be said to die of it, that, from the state of the lung, must not have died anyhow. This treatment by the cold bath, therefore, seems less applicable to pneumonia than to other febrile conditions where the structural defect is less ruinous. Yet in a suitable case, with hyperpyrexia and collapse and a limited amount of lung mischief, or with local symptoms favouring the belief that the lung was not breaking down, I think there is evidence enough in the cases quoted by Dr. Wilson Fox[2] (I have no personal experience) to justify the trial of the cold bath.

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[1] See Appendix B, 'Temperature.'

[2] Dr. Wilson Fox quotes three cases, two of which recovered. In the first the temperature rose to 110°, the patient (a woman of 49, with acute rheumatism and pericarditis) being insensible and apparently in the act of dying. The pyrexia was reduced by cold water and the application of ice to the body. After rallying somewhat, collapse was threatened and heat was applied, the patient being *largely stimulated with brandy.* The bath was afterwards repeated at 64, and subsequently ice-bags applied to the spine, but these on the whole failed of their object. In the second case (that of a man of 35), described as double pneumonia with pleural and pericardial effusion, the temperature rose to 107. Scruple doses of quinine were given. After the first bath subsequent threatenings of hyperpyrexia were treated with wet sheet packing. This patient *took as much as* 33 *ounces of brandy in* 24 *hours.* A third case (of acute rheumatism) with a temperature of 108·9, died. (See 'Treatment of Hyperpyrexia by means of the external application of cold,' by Dr. Wilson Fox, F.R.S.)

Over-activity in treatment.

There is a danger in all acute affections, and especially in pneumonia, where the attention is directed at once to two sets of phenomena, the local and the general, both of which are varying from day to day, of being needlessly active and restless in the matter of treatment, and following too closely with one's remedies in the track of the disease. So long as the pyrexial course continues equable, or with such variations only as are within the pattern of the disease, the extension of the local inflammation, even the implication of the fellow lung—grave as this is, no doubt, in its own bearing—does not call for any change in the treatment originally proposed. Even a very irregular course of pyrexia, or a temperature at the outset unusually high, does not necessarily indicate an unusual severity or duration for this disease. The symptoms for which the physician is to be especially on the watch, with a resolved mind as to his conduct when in face of them, are chiefly two, urgent dyspnœa and failing strength.

Convalescence.

Little need be said of the treatment of convalescence. Pneumonia differs from typhus in the little wasting it produces; it differs also in the same respect from alveolar catarrh, where, even in favourable cases, the loss of flesh is often rapid and considerable. Convalescence from true pneumonia is short and secure and little subject to relapse; that from alveolar catarrh is tedious and interrupted. While one leaves the patient pretty much as it found him,[1] the effects of the other will often linger indefinitely in the form of bronchial catarrh. Yet with the perfect recovery of the general health which is the rule of pneumonia, traces may remain of the bygone illness. Thus the affected side may continue dull to percussion from thickening of the pleura, or forced inspiration may give rise to redux crepitation from unfolding lung, or the respiration may be merely harsh and coarse, of different 'pitch' to that of the sound lung, and with a prolonged expiratory murmur. Inasmuch as these conditions are consistent with the perfect recovery of health, patients often pass out of sight at this period, and it is not easy to say how long such abnormal sounds may be expected to endure.

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[1] Nevertheless it happens sometimes, and especially with scrofulous children, that an attack of true lobar pneumonia is badly recovered from, so as to make it appear that the subsequent ill-health and wasting take their origin from the acute illness. This after all is no more than we see with other acute diseases with such subjects. There is no reason to dread pneumonia more than other affections for such children, nor is it often that lung disintegration either originates from, or is accelerated by, an attack of the kind. (See Chap. xiii.)

It is customary in this as in other affections to seek to speed the recovery by means of tonics of various kinds. The circumstances are such that almost any of these will be favourably received. Certain special 'indications,' which need not here be enlarged upon, may call for the employment of iron or bark or cod-liver oil. The natural course is towards recovery, and it is impossible to measure the aid which these afford to it.

The sum of the treatment.

Here, then, I may conclude what will appear, in comparison with the more robust therapeutics which the very name pneumonia still suggests, but a bald account of the treatment of a grave disease. It would not be difficult to be more explicit and more peremptory, for there is no absolute scarcity of remedies, nor is a timorous misgiving the characteristic of those that commend them. We may be sure at least of this, that as regards remedial measures pneumonia is destined to undergo vicissitudes. The treatment of disease is governed by other motives besides reason and experience, and men call that a refuge in danger which before they have avoided as a snare. Thus with the affection we have been considering, so long as the gross results are not disastrous we represent nature as beneficent and charge ourselves with the duty of aiding her efforts. But such a view does not embody a necessary truth, and in different circumstances it would be easy to change the figure and revert to the old notion which regards the operations of disease as those of an enemy who is always to be opposed and may sometimes be out-manœuvred. The treatment that proposes to make drugs subordinate, to trust in the sedulous care of a nurse and judicious feeding no less than in the pharmacopœia, would fade away suddenly in the presence of a high death-rate. We should appeal then to the ascertained influence of tartar emetic and the immediate efficacy of early bleeding. Multitudes would never be suffered to die untreated; the look-out of the expectant treatment would be altered. At present, as it happens, we are content with the adoption of means which have the advantage of being eminently reasonable, as being based, not on the shifting sand of to-day's therapeutics, but on a wider application of such means of maintaining vitality as the whole experience of mankind is united in recognising.

APPENDICES

APPENDIX A.

THE MODE OF PRODUCTION OF CREPITANT RHONCHUS.

Crepitating rhonchus or 'minute crepitation' may be exactly imitated, as Dr. Williams first noticed, by rubbing a lock of one's hair between the finger and thumb; it may be imitated, too, less closely, by the cracking sound of salt thrown upon the fire; both these resemblances being purely accidental, and indicating no corresponding similarity of mechanism. But crepitating rhonchus may be imitated, moreover, by crumpling within the palm fine tissue paper. The likeness is not striking at first, but when the fingers have learnt to exercise a correct pressure, and the paper is neither too tightly nor too loosely crumpled, its crackling may be brought nearer and nearer to the lung sound. Similarly, if a small piece of moistened animal membrane—the skin of a sausage or poloney—be rolled up and squeezed between finger and thumb, it also (of the proper moistness and with the proper pressure) will *crepitate* like the inflamed lung or the rubbed lock of hair. Thus, though the true crepitant rhonchus is frequently confounded with other sounds, this is from no want of kindred sounds for comparison. The best of these is, with most of us, continually within reach of the fingers.

Yet, while the resemblance obtained from the lock of hair is sufficiently accurate even to satisfy Dr. Walshe, the hearer is not unaffected by the knowledge that this is a 'rubbing sound,' while the morbid one is not. So long as the mind is alive to this real difference the ear is slower to appreciate the mere sound likeness. We seek a sound with which to compare minute crepitation which shall resemble it altogether, in mechanism as well as in tone.

I have said that nobody knows for certain what this mechanism may be. The theory that the crepitation is caused by the passage of air through liquid of a certain consistence contained in the air-cells is completely upset by the observation that the sound occurs with fluid of *any* consistence ; nor can any reason be alleged why, upon this hypothesis, there should be a crepitus with the entrance of air but none with its exit. Why should the rhonchus be inspiratory only? The presence of fluid accounts for a noise, but not for this noise. A later theory refers the sound to the repeated unsticking of the air-cells with every act of inspiration—an unsticking which must take place, as Dr. Waters points out in his admirable lectures, in the air-sacs themselves, since the finest bronchules may be found unaffected. To this view there are many obvious objections which need not be dwelt upon, for the single observation of Dr. Walshe that 'crepitant rhonchus of typical perfection may occur with the purest watery œdema,'[1] is of itself absolutely fatal to it.

If, then, neither bubbling nor sticking is at the bottom of this sound, what remains? May it not be that it is produced by an agency strictly analogous to that which will cause a similar sound out of the body—not the rubbing of the lock of hair, or the explosion of salt, but the squeezing of moistened animal membrane?

It will be admitted that the condition of lung—be it pneumonic or not—which is essential to the production of crepitant rhonchus is a condition of œdema ; that the air-sacs, and the air-sacs alone, contain fluid. Yet in pneumonia, probably, this effusion will not be equally distributed : some cells will be full of it, others less full, and some empty and even over-dry. Moreover, the consistence of the exudation will be watery in one part, more viscid in another, and in some places beginning to coagulate. This is in accordance with the pattern, not, indeed, of all, but of most, pneumonias that we come to see after death; the process advances unevenly, so that various stages of inflammation are to be found side by side in the same lobe.

What should be the effect of an inspiration upon such a lung?

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[1] For a concise and able statement of the hypotheses on this subject, and the objections to each, see Dr. Walshe 'On Diseases of the Lungs,' p. 110, fourth edition.

By that act air would be brought into such cells as were yet unoccupied, while at the same time the adjacent and loaded cells would be subjected to an increasing pressure, so as at length, with the deepening breath, to produce a noise by their oozing. The pressure arises from the fact that fluid is occupying air space. The volume of the lung and the force of inspiration remaining unaltered, this pressure would be proportioned to the amount of air proper for aëration which was thus excluded. The precise circumstances of the living organ cannot, of course, be reproduced after death, but by pinching any œdematous lung it will crepitate.

The essential characters of this rhonchus consist with such an explanation. They are these : It occurs with inspiration only ; when first observed, and again when about to depart, it is heard only at the end of inspiration ; the deeper this inspiration the more distinct is it ; when it has persisted for a longer time than usual it becomes audible, *sometimes,* with expiration as well as with inspiration, and may even be louder with the latter ; occasionally it is absent altogether from first to last. All this accords with the hypothesis. At first, while the fluid accumulation is inconsiderable, it is only with a full inspiration that the pressure reaches it, or, rather, that it is sufficient to produce that oozing of fluid upon which the sound depends. The rhonchus, therefore, is either not heard at all, or heard only at the end of inspiration. Soon, however, the fluid increases, and then the entrance of air is immediately followed by morbid sound ; the rhonchus is heard from beginning to end of the inspiration. But presently, as the exudation increases in quantity, it alters in its physical character. It becomes more and more viscid, and at length wholly or in part consolidates. It is now no longer squeezable, or, if squeezed, it requires a greater pressure before an oozing sound is obtained from that portion of the exudation which still remains fluid. Accordingly the rhonchus, if audible at all, is audible now only at the end of inspiration. It reverts, that is, to the character it had at the beginning, although the same physical conditions do not return.

And how comes it that when this crepitant rhonchus has existed for some time it begins to be heard—in certain cases, not always—with expiration as well as with inspiration ? The hypothesis would account for this also. It is known that when a fluid exudation has occupied the pulmonary alveoli for a longer period than usual, the alveolar walls themselves become affected by reason of their contents. Through continued soaking with a fluid of the proper consistence, they swell out and eventually stiffen. Such lungs are described as exhibiting a notable loss of elasticity. When brought to that condition the lung-tissue may not only emit the particular sound in question when squeezed (that is with inspiration), by virtue of the fluid that it holds, but, furthermore, when the pressure is removed and the fluid again distends the air-sacs (that is, with expiration), the stiffened walls *creak* as they move. This expiratory rhonchus is, then, both more obvious than the inspiratory and of coarser quality, just as a wet sponge creaks loudest, not when it is pressed, but when the pressure is relaxed.

No doubt some of the phenomena of crepitating rhonchus are satisfied by the prevailing theory—that, namely, of a sudden and forcible expansion of tissue glued together ; but they agree still better with this one. The entire absence of this kind of crepitation in some rare cases of pneumonia is probably due to the very rapid solidification of the exudation. There is neither bubbling nor unsticking nor oozing ; the vesicular respiration is suddenly abolished, and in that part of the lung whence this sound comes there is silence. Admitting that the seat of the rhonchus is the air-sacs, this is intelligible upon all the hypotheses alike. But why is minute crepitation absent when there is no such rapid consolidation ? Why, especially, is it absent sometimes in pleuro-pneumonia, where pleuritic stitch is so acutely painful that the patient fears to fetch the breath he so urgently needs ? It can always be said that the sound is out of reach. But suppose a large portion of lung to be implicated, yet not solid. It cannot be maintained that an individual so circumstanced (I am quoting what I have seen) could by any exercise of caution exclude the air from a particular district of his lung ; its murmur, indeed, is to be heard here as elsewhere. But it can very easily be maintained, where the pain of inspiration is excessive, that a man may so far command himself as to stop short of a full breath, dividing his troubles within certain limits, in his own way, between dyspnœa and pain, and that this bated breath would fail to furnish the pressure requisite for crepitation.

We know, indeed, what can be done, in the way of sound, by the mere unfolding or unsticking of lung. What else is the so-called ‘ redux crepitation ’? The peculiar *gush* of this sound, its occurring for a few times only with forced inspiration, and ceasing abruptly, for that while, so soon as the lung has become fully inflated, are characters so marked and special that the student must often regret the very trivial importance of a sign so easily recognised. It will certainly never remind him of minute crepitation.

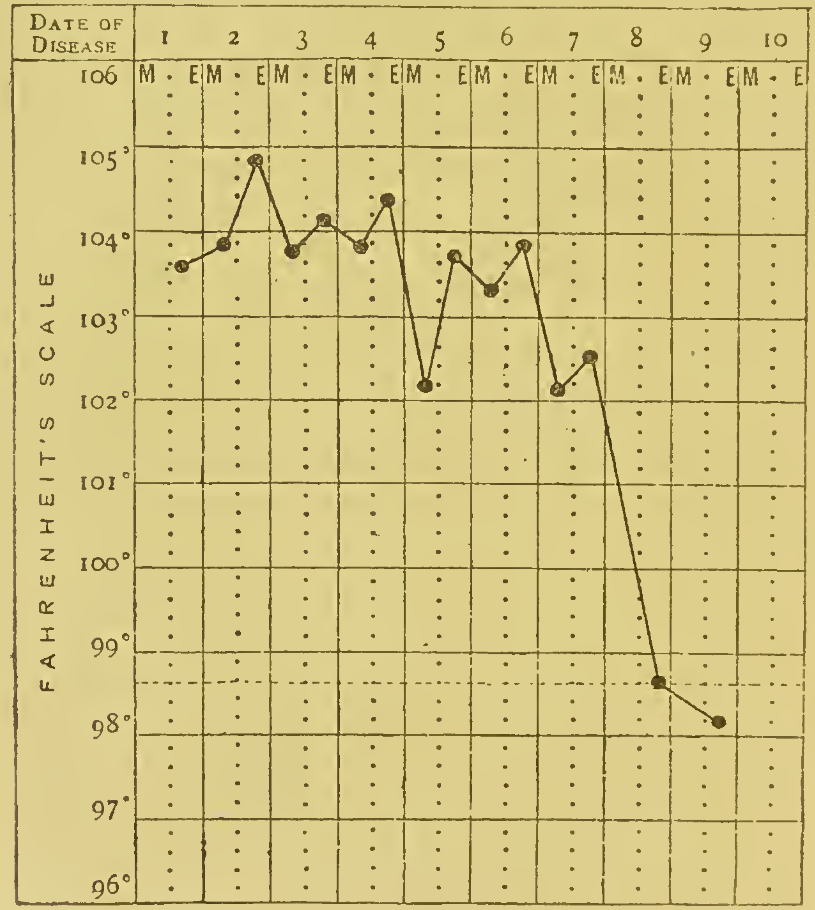
Perhaps the objection will be made that crepitating rhonchus is a 'dry sound,' while the sound of oozing is emphatically a moist one. If this be so, all the theories are faulty alike, for all agree in recognising the agency of fluid. And inasmuch as the lung which yields the sound is not drier but wetter than in health, it seems not improbable that this 'sensation of dryness' is excited by moist material, and, therefore, like many other sensations, delusive. For my own part, I would rather side with those who defer offering descriptive adjectives for this sound until such time as clearer notions are obtained as to its mode of origin.

More might be noted. The character of this rhonchus in infancy, its transition into the obviously 'moist form,' the delay which is sometimes noticeable in its occurrence, and the harsh respiration which immediately precedes it, are all points to be dwelt upon in favour of this theory. It is unnecessary. If my view be the true one, it will assert itself. In any case, the fact that prevailing theories upon the subject are admittedly untenable is a sufficient excuse for suggesting another; nor does it seem to me unreasonable, having regard to the physical state of the part where this rhonchus is produced and the essential characters of the sound, to have recourse to this agency of pressure, and thus to bring post-mortem crepitation and living crepitation within one category.

APPENDIX B.

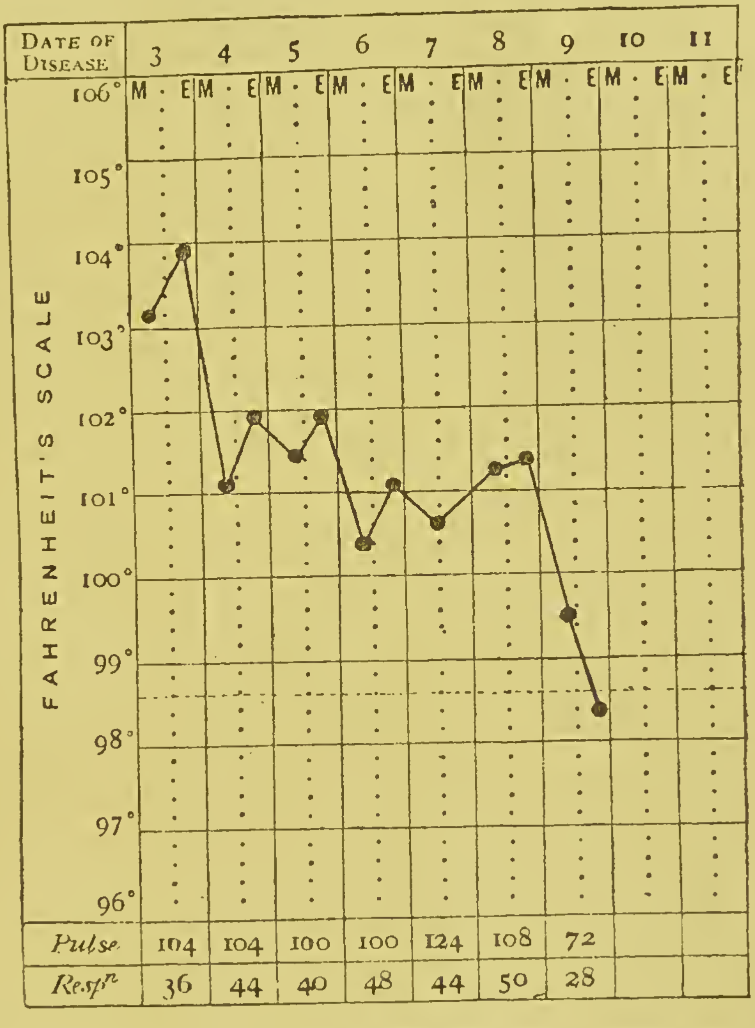
RANGE OF TEMPERATURE IN PNEUMONIA.

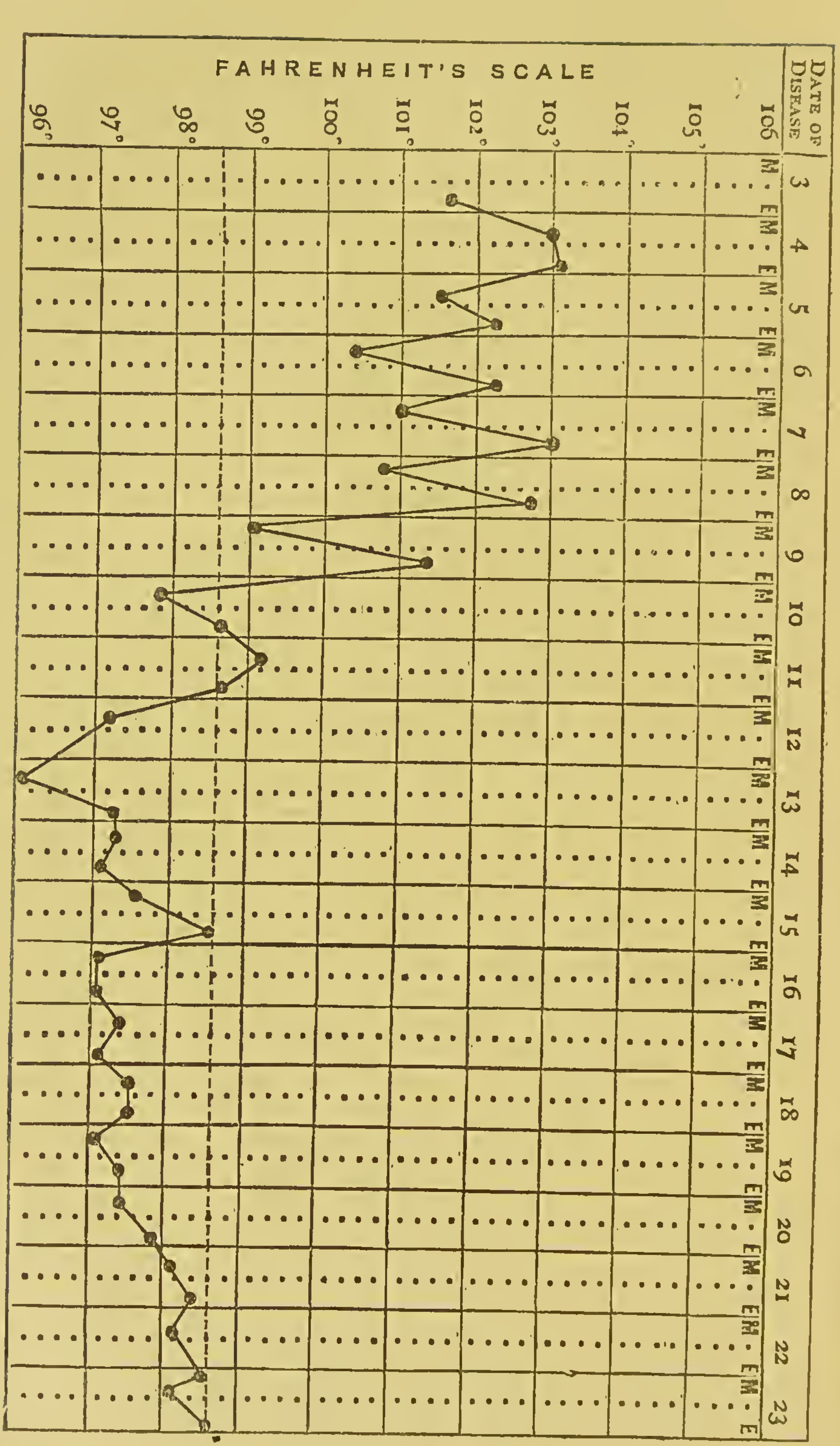
I CANNOT discover that the temperature of pneumonia, of which Wunderlich's typical tracing is given below, has such peculiarities as suffice to distinguish it from other pyrexia. When it is said that the pneumonic fever exhibits early a high—often its highest—temperature, that the same or a similar heat is maintained until the



fever suddenly departs towards the end of the first week, and that the morning temperatures are usually somewhat below the evening, I think all is said that is commonly true in regard to this particular feature of the disease. But as much may be seen with the pyrexia of other affections, of typhoid for instance.

Of the following temperature-charts, on this page and the next, which is which? On comparing the two it will be seen that there is about the same difference between the morning and the evening register in both, and that in degree and time of endurance the pyrexia is such as we are told to expect in pneumonia; in the chart on p. 250 there is an anticipation the day before of the final crisis, and after it the temperature sinks below 98°, which is according to the 'typical average' of the same disease.




FAHRENHEIT'S SCALE
96°
97°
98°
99°
100°
101°
102°
103°
104°
105°
106
DATE OF DISEASE
3
4
5
6
7
8
9
10
11
12
13
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16
17
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M
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Now this latter chart represents typhoid in a child under my care at the Hospital for Sick Children, quite distinctive, but never severe, and with no lung affection. That on page 249 was pneumonia in a man of thirty,[1] a patient at the Westminster Hospital, serious for a while, but undergoing critical change on the tenth day. Contrast with these Wunderlich's typical range of temperature (from Thermometry, fig. 51, Table VI.), and it will be seen to have features in common with both.

Without venturing to insist on a typical range of temperature for pneumonia, therefore, I think it is not untrue to describe it as giving high temperature at the beginning, and, between that and the end of the first week, keeping pretty even as between the morning and evening (the latter being always the higher), yet more liable than typhoid to sudden elevations and depressions, while after the first week, crisis or not, its range is sometimes very irregular.

High temperature is occasionally seen to persist, as has been mentioned, where in other respects the phenomena of crisis are fulfilled. This is accounted for by Dr. Long Fox, on the supposition that the pleuritic inflammation outlasting that of the lung, exhibits a pyrexia of its own.

A high temperature at the beginning of pneumonia does not *ensure* a severe attack ; a sudden elevation may be ephemeral, and does not always *imply* an increase of the disease ; a stationary, or even a falling temperature, *may* concur with a rising pulse and increased frequency of respiration ;[2] one patient may die with a temperature never exceeding or reaching that of another whose attack has given no occasion for anxiety.[3] It would neverthe-

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[1] To the chart on p. 249 the record of pulse and respiration has been added, as affording an illustration of the variable relation sometimes maintained by these in pneumonia.

[2] Thus Dr. Peacock reports a case of pneumonia fatal on the sixth day, and examined after death, where the highest temperature, 102·5, was reached on the fourth day, from which time till death it fell rapidly, just as it might do in a favourable crisis, reaching 97·6 very shortly before death. (Peacock's 'Report on Cases of Pneumonia,' in 'St. Thomas's Hospital Reports,' vol. ii.

[3] As I write, Dr. J. G. Bacon of Saratoga reports to the 'New York Medical Record' as follows :

'Thinking a brief statement of two cases of pneumonia, which I have still under treatment (though now convalescent), might interest your readers, as regards temperature, I submit them. The first case, a girl aged 16 years, strong and full-blooded, had pneumonia of the left lung. Temperature began rising up to fifth day, when it stood, as carefully noted by a self-registering thermometer, 107·5. The sixth day it fell to 104° by evening. The condiless be far from the truth to say the thermometer is of small use in pneumonia. But it is not a certain guide, and must never be trusted alone. One of its chief services in diagnosis is to mark the difference between this disease and other forms of lung-consolidation. It may be added that erroneous temperature-registers are not uncommon owing to the use of uncertified thermometers.

tion of the patient otherwise showed no cause for alarm, as far as consultation could decide. She is now about, and free from cough, and gaining her strength fast. The second case was her brother Frank, aged twenty years. He was seen twelve hours after he began to complain. Pulse 165, wiry. Slightly delirious. Examination revealed pneumonia of right lung. Temperature, very carefully taken thirty-four hours after I called, was 110°. I could not believe my eyes until I had repeated the experiment several times with the same result. The expectoration was nearly pure blood for forty-eight or fifty hours; cough harassing. Now, here is a strange feature (to me) in this case: the temperature was 110° at 5 P.M., and the next morning, at 9.30 A.M., it had fallen to 99°, or rather, 98·08°. An intense diaphoresis occurred, which continued for twenty hours.'

APPENDIX C.

ILLUSTRATIVE CASES.

THE following narratives are intended to illustrate some of the leading features of pneumonia, including certain incidents of the disease, more or less common, to which reference has been made in the foregoing pages.[1]

CASE I.

Pneumonia of lower lobe, left lung—Ascribed to exposure—Crisis on the sixth day marked by sweating—Pulse during convalescence becoming remarkably slow.

Stephen B., aged twenty-two, admitted into Westminster Hospital Feb. 21, 1871. Three days before, while crossing Westminster Bridge (the day being cold and gusty) and in perfect health, he was suddenly seized with 'stitch'; he at once took to his bed, and was carried from it to the hospital.

On admission he has the dusky florid look and pained expression of pneumonia; the sputa are rust-coloured, but as yet very scanty; a little unmixed blood is also spat up; pulse is 108, temp. 102°·2. The lower part of the left chest is dull to percussion, and yields increased vocal fremitus; breathing in this situation is purely tubular. At the upper part of the same side, above the dulness, there is crepitant rhonchus.

On the 23rd, (or sixth day of his illness), the pulse had risen to 120, temperature was 102°·1. There was no urgent symptom. The man was now sweating profusely.

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[1] In noting some of the following cases I have been much assisted by my friend and colleague Dr. de Havilland Hall, Medical Registrar at the Westminster Hospital.

On the 24th, (the fourth day from admission, and seventh of illness,) the pulse was 93, or 27 below the previous day, and temperature 101·8.

On the 25th redux crepitation had nearly replaced the tubular respiration ; the man was as good as convalescent ; the pulse 72, temp. 99·2.

The pulse, continuing to fall, became at last remarkably slow ; on March 1, eleven days from the first seizure, it was 48, when the patient was practically well, although some dry cough lingered.

The above sketch illustrates in outline some of the leading features of ordinary pneumonia, as described in Chapter iii. Its apparent origin in exposure, its sudden and severe access, its crisis, equally sudden, about the sixth day, followed by sweating. The fall of the pulse in this instance was much more marked than that of the thermometer. And (as occurs sometimes in continued fever also) the pulse-rate succeeding the pyrexia was abnormally slow. The case did not call for stimulation, and did perfectly well left to itself.

CASE II.

Pneumonia of right upper lobe—Rapid development—High, variable temperature—Marked crisis on the fifth day—Estimation of urea during crisis—Short convalescence.

George W., twenty-one, labourer, admitted to Burdett Ward, Westminster Hospital, June 7, 1875.

A healthy, stout-built man, addicted to some excess in beer, till within a few months, when he became temperate. He was at work and well till the evening of June 3, when he went to bed and slept as usual. At six o'clock the following morning he complained of stiffness, pain in his legs, and headache ; he had a slight rigor and vomited. Within half-an-hour of the commencement of these symptoms, cough set in, and along with it blood-coloured expectoration. He had also pain in the right side, but this did not appear to be acute.

When seen on the third day of his illness, he had a flushed anxious face, and lay, not evenly on his back, but with the left or affected side raised above the level of the other, a position which he maintained throughout. Breathing was extremely rapid and panting, the nostrils being kept in rapid action. With a pulse of 108 only, and no marked 'stitch,' the respirations reached 72. Cough was short, not harassing, sputa scanty, and of the characteristic rust-colour. Over the upper half or less of the right lung the respiration was bronchial, approaching tubular; there was diminished resonance posteriorly, *and no crepitus.* The tongue was moist and clean, but the man had no desire for food. He did not now complain of pain.

On the morning of the day of admission (being the third of illness) the temperature was 103°·2, but in the evening it rose to 105°·2, falling again towards midnight to 104°.[1] On the following day (June 8) the physical signs were more marked anteriorly, the infra-clavicular region yielding marked dulness, and tubular breathing audible both there and posteriorly, with other phenomena of solid lung. The patient's aspect was unchanged; he had wandered in mind through the night, but had no active delirium. The motions were dark and rather loose, sputa still rust-coloured; urine contained only a trace of chlorides, was free from albumen. *The pulse was* 96 *and the respirations* 60. The morning temperature of this day was 102°·8; in the afternoon it had risen to 103°·3, and the same night to 105°.

At the commencement of this second temperature rise (that is to say, at two o'clock in the afternoon of the fourth day of pyrexia), it was directed that the urine passed during the next twenty-four hours should be collected for quantitative estimation[2] of the urea eliminated. [It will be seen directly that in the course of this collection crisis took place.] The total urine so passed (none having been lost) was 1,130 cubic centimetres, of specific gravity 1020, percentage of urea 3. The total amount of urea eliminated in the twenty-four hours 33·9 grammes (a quantity hardly above the normal). Of chlorine excessively minute trace only.

Meanwhile the patient fell into a deep sleep, which was broken only once or twice when he roused himself to pass water. On the following morning, when still in a state of lethargy, the pulse was 72, respirations 44. The temperature in the early morning was 103°, or just two degrees lower than the previous night, and continued rapidly falling. In the forenoon it had sunk to 99, and by midday had subsided to 97·3, or more than a degree below the line of normal heat. In other words this man's critical change, which concurred with a normal urea discharge, was accompanied by *a fall in temperature within little more than twelve hours of more than seven degrees Fahrenheit.* With these marked phenomena the aspect of the patient corresponded; and the sudden change from the fever flush, and anxious watchful aspect to an expression of mere drowsiness from loss of sleep, was very striking.

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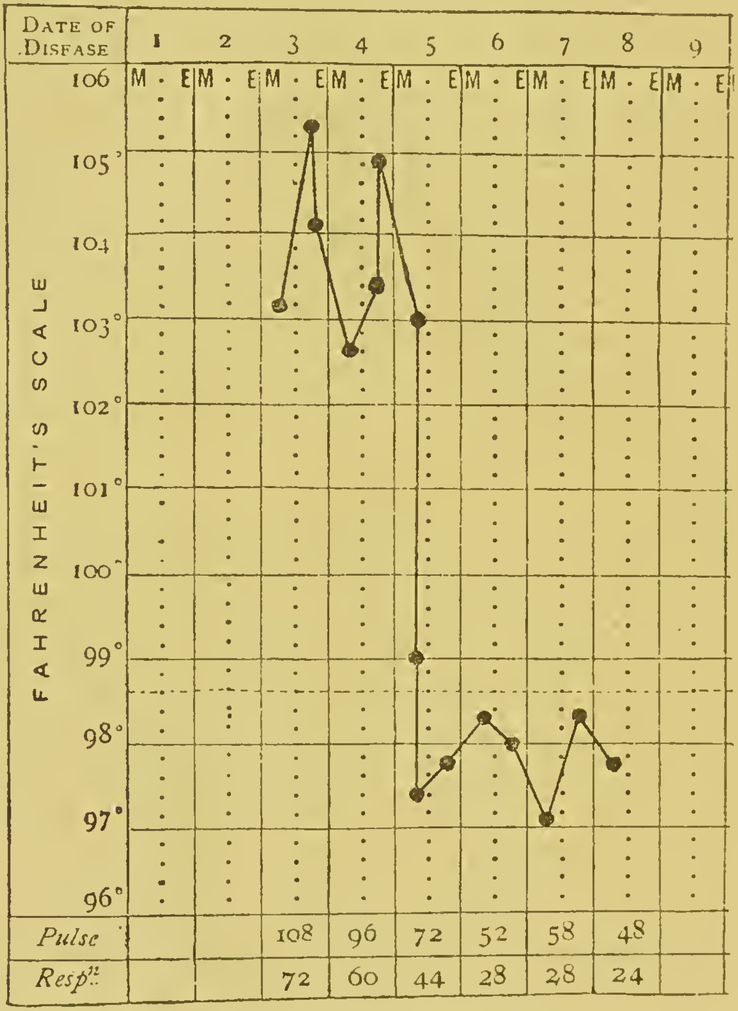
[1] See Chart temperature appended.

[2] For the greater assurance of accuracy, my friend and colleague Dr. Dupré, F.R.S., lecturer on Chemistry at the Westminster Hospital school, was kind enough to undertake this analysis.



On the 10th the sputa were quite uncoloured, watery and spittle-like; respiration at the upper part of the right lung was still bronchial, and some coarse (redux) crepitation was now heard for the first time. The man was now drowsy and contented. The temperature had risen, but was still slightly below the normal point; pulse was 52, respirations 28, appetite returning. For a day the pulse was below 50. In ten days he was up. Treatment there was none. The man took no alcohol until his convalescence was established, and then only a daily glass of beer. The mixture of acetate of ammonia formally ordered was never rigidly insisted on, and soon discontinued. He hardly lost flesh in the attack, and it was not necessary to seek to hasten his recovery with any medicinal appliance.

The case shows that early high temperature does not imply prolonged or dangerous attack; that the affection of the upper lobe has not necessarily special phenomena of its own; that the amount of urea elimination is not necessarily either in excess or defect at the time of crisis. It also shows from the Temperature chart that the ratio between pulse and respiration varies considerably from day to day, and that a quickened respiration may correspond with a lowered pulse and lowered temperature.

The final issue suggests certain reflections in the matter of treatment. What if this man, when breathing sixty a minute, had been bled? What if alcohol had been given at the same point, or large doses of quinia? Could the bleeding or the drugs have escaped the credit they would not deserve?

The following abstract (though the notes are too brief), while it serves to illustrate some points in which pneumonia may be imitated by lung congestion, has an interest of its own.

CASE III.

Pericarditis of obscure origin—Physical signs of engorgement simulating pneumonia—Fits of syncope- Death by syncope—Post-mortem examination.

Robert H., aged twenty-eight, whitesmith, admitted into Burdett Ward October 27. He has been subject to chronic rheumatism (so called), but had never suffered an acute attack.

Two weeks ago, when under treatment as an out-patient for pain in the chest, supposed to arise from dyspepsia, he was seized with sudden dyspnœa and acute pain referred to the epigastrium. The pain subsiding, had left him still breathless and unequal to exertion.

On examination (the pulse being just over 100, respirations not greatly hurried, the man raised in bed and with an anxious face), dry rubbing was distinctly audible over the base of the heart. Over the lower half of the right lung was increased vocal fremitus, with small inspiratory crepitus. The left lung yielded bronchial respiration and marked ægophony, and on the second day small crepitus became audible in it also. During the patient's week's residence the pulse was mostly under 90, and the temperature, at first 102°, fell to 99°.

On November 2, after slight exertion, he had a fit of sudden syncope (angina-like, but without its pain), and was thought to be dying or even dead ; but, after artificial respiration, revived.

The physical signs did not materially alter, save that the pericardial friction became for a while louder and more grating, and then (that is after the syncope) finally disappeared.

Three days after this first fit the temperature again rose to 102° ; crepitus was still audible at the base of the right lung, but with this absence of pericardial friction, no sign of effusion in the pericardium was made out.

The following evening, November 6, a second attack of syncope occurred, and artificial respiration was again employed with success, the patient being shortly restored to his before condition. On the morning of November 7, however, a third fit seized him, in which he speedily died, although the same means as before were resorted to for his restoration.

It may be allowable to interpose a few words between this imperfect sketch of the case and the report of the post-mortem inspection.

The origin of the pericarditis was obscure ; there was neither rheumatism nor uræmia to account for it. Admitting the fact, however, upon the evidence of the rubbing, how was the pulmonary engorgement related to it ?

That it was commencing pneumonia might be suggested by the character of the crepitus, by the temperature of 102°, and by the known association of pericarditis with that disease.

On the other hand, not only were most of the characteristic symptoms of pneumonia absent, but the physical signs, such as they were, did not progress, and they indicated a symmetrical implication of both lungs, the always-to-be-suspected 'double pneumonia.'

In fact the progress of the case, while it soon disposed of the theory of pneumonia, brought new phenomena to bear upon the diagnosis. The syncopal attacks, which had all the essentials of angina, might account, upon the principles already discussed, for pulmonary hyperæmia. A weakened circulation, due to fatty degeneration, would explain everything save the pericarditis. Starting from that, the rest was easy. That a heart showing signs of failure should be still further embarrassed by inflammation of its pericardium, it was easy to suppose; what remained was to explain the pericarditis. This task was not achieved during life; but post-mortem inspection at once removed the obscurity.

Post-mortem Examination.—All the abdominal organs were healthy.

Both lungs were highly congested, and semi-solid towards their bases; the right adhered by oldish bands to the costal pleura. The left pleura contained some fluid.

On opening the sac of the pericardium it was found stuffed with currant-jelly blood-clot, with streaks here and there of uncoloured (ante-mortem) clot. The surface of the heart exhibited patches and bands of lymph in the place and manner characteristic of recent pericarditis, but tinted with the blood colouring. The visceral pericardium was thickened, as from a previous attack of pericarditis.

This condition, and the symptoms during life (the repeated syncope, and pericardial rubbing suddenly abolished after the second fit) received explanation from the discovery of a ragged rent just beyond the aortic semilunar valves in a dilated portion of the ascending aorta. Through the rupture of this small aneurism blood had oozed, at first sparingly into the pericardial sac. Pericarditis was thus set up, while the impaired action of the heart led to passive lung congestion. Presently, by the gushing out of more blood as the rent became larger, the friction sound was lost, and at last by a final outpour the fatal syncope was brought about and the end accomplished. (See Chap. x., pp. 138 et seq.)

CASE IV.

Pneumonia of left apex—Wild delirium—Insensibility—Sudden amendment on the ninth day—Rapid recovery.

Elizabeth T., twenty-nine, married woman, with no phthisical history in her family and herself hitherto perfectly healthy, was seized with violent ear-ache on July 29 at 8 A.M., having arisen feeling quite well. In three hours some discharge commenced from the right ear, with immediate relief to that pain, although general headache continued. The same evening pain commenced in the limbs and left side. She was unable to lie on this side, or to take a deep breath. On August 2 slight shivering occurred, and she was admitted into hospital the same afternoon[1] (the fifth day of illness).

The pulse was now 152, small, and respirations 48; the temperature 104·2°. On this day no abnormal sounds could be detected either in lung or pleura. On the next morning, however, breathing was tubular at the left apex, with dulness on percussion anteriorly reaching to the third interspace. There was also a little fine crepitation. Slight cough, with sputa viscid and rusty, pulse 144, respirations 48. In the urine was no albumen, but marked deficiency of chlorides. It is not to the purpose to enter here particulars of the progress of the physical and other signs for the two following days. It is sufficient to say that consolidation of the upper portion of the left lung was very clearly indicated, that the pulse remained over 140, with respirations varying from 50 to 72, and temperature a little over or a little under 104°, the patient being quiet and not delirious, her tongue moist and furred. She was given ammonia and chloric ether, and three ounces of brandy daily.

On the morning of August 5 (being the eighth day of illness) this woman became somewhat delirious; her pulse was at this time 146, and respirations 72; the area of dulness had not extended, and coarse inspiratory ('redux') crepitation was now heard. As the day drew on the delirium deepened, and in the afternoon the patient began to rave, throwing her arms about, beating the bed, refusing all nourishment, and wasting her little strength in piercing yells. The pulse was now flickering, and hardly to be counted; the respirations were no less than one hundred; the eyes became fixed and upturned, with *total insensibility of the conjunctiva.* The features were now pinched ; tongue and skin dry and parched ; death seemed impending. With great pains she had been got to swallow by drops nearly half an ounce of brandy. She was now—when seemingly dying—ordered an enema of eight ounces of beef-tea with half an ounce of brandy, only a part of which was retained. Upon this some improvement appeared in pulse and feature, but delirium of the same wild kind as before was not absent for long. The enemata were repeated at intervals. Towards midnight the respirations varied between 72 and 90, and the pulse improved in strength. Owing to the patient's violence and restlessness it was impossible to take the temperature accurately. On the evening of the 4th it was 104°, and it is believed that it did not afterwards exceed that point.

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[1] This case and the next were admitted under the care of Dr. Basham, who kindly consented to my observing, and making this use of them.

Fifteen minutes after midnight on August 6 (the beginning of the ninth day of illness) this woman suddenly recovered her senses and recognised her husband. She now accepted nourishment readily. The pulse fell to 120, respirations to 60, and temperature to 100°; a fall of over 3° from the previous day. The urine, which had been retained for some hours, was now passed in large quantity. When left to herself the patient would fall into a muttering delirium, from which she was easily roused. The skin was now perspiring. She shortly fell into a sound sleep, and with this it may be said that her peril ended. Later on of the morning of the 6th, when the pulse was 124 and very feeble, and respirations 44, the body freely sweating, the temperature was still 100 ; it fell *continuously* throughout the day, and was 99° on the morning of the 7th. The sputa were now yellowish and without blood-stain. Signs of consolidation of lung were still marked. On the 7th some redux crepitation was heard, and by the 9th dulness had almost disappeared.

It is unnecessary to pursue the case in detail. After the 8th the temperature remained normal ; the pulse and respiration fell together, although the former remained over 100 until August 10. On the 16th, being just three weeks from the earliest symptoms, and ten days from the crisis, the woman was able to get up for a while, and shortly after she went out well.

This case (which is remarkably similar to the two which are to follow) suggests many reflections upon points alluded to in the text. It is also open to the same remarks as the example before it. What credit would not have been given, and what would have been really due, to any exceptional measures, as bleeding, or ice-cold water, or aconite, or digitalis, adopted in so grave an emergency. I would, moreover, draw attention to the illustration here afforded of high fever with profound implication of the nervous system, along with pneumonia of no great extent. The gravity of the affection is no more measured by the lung disease than is the gravity of typhoid measured by *its* local part in the bowel-ulceration. True, the local malady may in either case bring death, and has always a danger of its own, but with a strict limitation of such local malady the patient is not made safe; he is exposed to other perils in the way of his disease than those which obviously confront him from the condition of a particular organ.

CASE V.

Pneumonia of upper lobe—Active delirium—Late appearance of physical signs—Lividity and symptoms of collapse on the ninth day along with defervescence—Crisis on the tenth day.

John A., aged seventeen, admitted March 19, 1875, having been taken ill on the evening of the 17th with shivering, giddiness, and nausea, and delirious during the same night. When examined on the 20th he had a pulse of 130, and temperature 104·6; tongue coated, his condition generally resembling fever. He wandered when left to himself, but answered questions rationally. The chest-sounds were noted as normal. The same night he became actively delirious, and some *fine crepitation was now first audible* (without dulness) over the upper third of the right chest. On the 21st (or fourth day of illness) the physical signs were more marked; dulness at the right apex, tubular respiration, the heart's sound being transmitted with great clearness through the solid lung. He had now rusty sputa. During the next three days, from March 22 to 25 (the eighth day of illness) the patient continued violently delirious; temperature 103·6, rising (on the 24th) to 105·2; pulse 130, full and hard; respirations from 68 to 76.

On the 26th (the ninth day) with a falling temperature (101°), and pulse still 130, he was, as to his general condition, markedly worse; his face was now livid and pupils dilated, the tongue was dry and parched, and he sweated profusely. No urine had been passed during many hours, and the accumulation in the bladder had to be withdrawn by catheter. The violent delirium had given place to mere prostration. He had profuse sweating, was barely conscious, and the motions were passed in bed ; he appeared to be dying. He had been given four ounces of brandy from the first ; the quantity was now doubled, and the ordinary means of stimulation were employed.

At 3 A.M. of the 27th, having passed water naturally some hours before, and had some diarrhœa, this youth fell into a deep sleep (which the nurse believed to be his last) ; he awoke, however, at 9 A.M., greatly improved, with a temperature 99·4, clean and moist tongue, and sputa but slightly blood-tinged. The pulse was now 112, and respirations 50. Two days from this the pulse was 84, while the respirations were 32. The patient had no bad symptom from this time, and was convalescent a week later.

To the two cases just related, tending to show a special implication on the part of the nervous system as characterising apicial pneumonia, I would add one alluded to on page 67, and already published in the 'St. George's Hospital Reports,' vol. i.

The patient was a nurse-maid, aged twenty-eight. She had been taken suddenly ill with rigor, cough, and some dyspnœa six days before, and her friends had noticed at the same time a strangeness in her manner, but no actual delirium. There was nothing in the aspect of the patient (unless labial herpes be so considered) to indicate the nature of her disease. There was found, however, on percussion, marked dulness below the right clavicle, with which corresponded tubular breathing and brassy ring of the voice ; (temperature is not recorded;) the pulse was 112.

On the morning following her admission this girl sprang out of bed and ran naked through the ward. From that time, for four days, she continued actively delirious, making grimaces and talking wild nonsense. At the end of the fourth day (the usual lung symptoms remaining in abeyance), signs of sinking appeared ; the tongue began to get dry, and sordes formed about the mouth. But now of a sudden, the pulse, which had hitherto been 112 or thereabouts, fell to 70, and its rate did not afterwards exceed 80. Meanwhile the active delirium subsided, and the woman fell into a bewildered, puzzled state of mind, as of one just awakened. From this she very gradually returned to her natural manner. At the same time the disappearance of the morbid sounds at the lung's apex, and their replacement by perfectly healthy breathing, occurred in remarkable harmony with the general progress towards health. The patient was finally discharged in all respects well.

CASE VI.

Pneumonia of lower lobe, right side—Hæmoptysis, bloody sputa—Chlorides not diminished—Slow pulse—Crisis on the sixth day—Bronchitis supervening—Convalescence accidentally delayed.

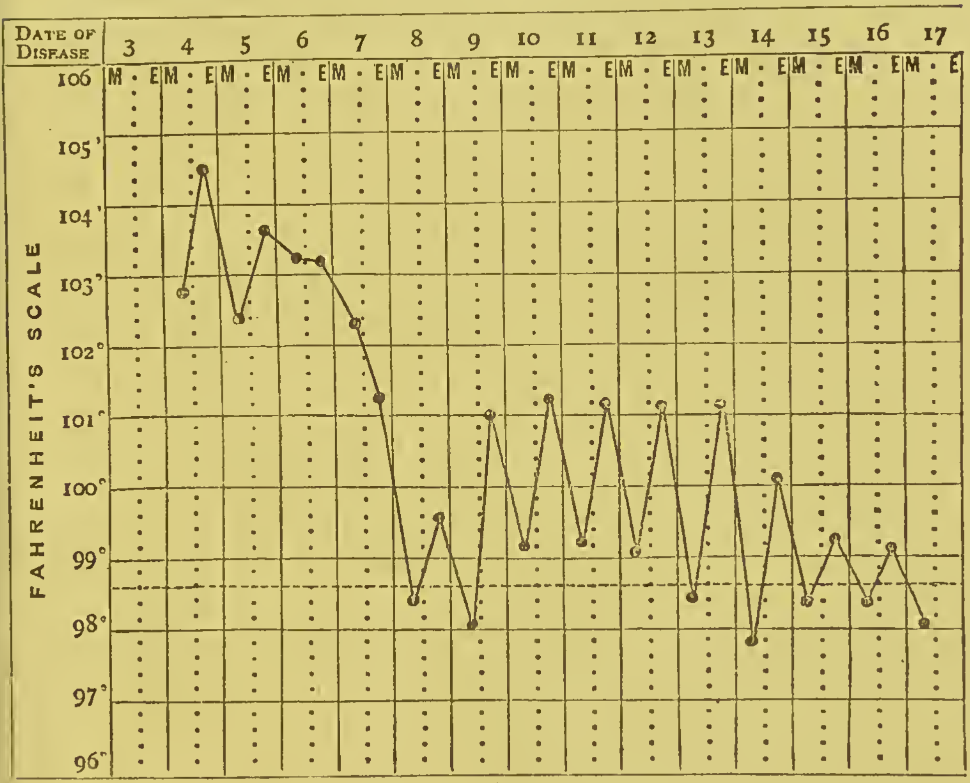
Henry B., aged twenty-four, lighterman, admitted July 1, 1875, into Burdett Ward. He had had rheumatic fever ten years before, uninterrupted health since, is of temperate habits. He was quite well up to and including June 27 (Sunday) on which day, in thinner clothing than his working-dress, he took an excursion into the country. (The season was ungenial, and after much enquiry this change of clothes had to stand for cause of the illness which immediately followed it.) Early in the morning of the 28th, after a good night, he was seized with pain across the right chest and in the forehead, but without shivering; he managed to get through two hours' work and then gave in, fairly beaten. The same evening he spat up 'congealed blood.'

On admission early on the fourth day of illness, his face was flushed, aspect anxious, decubitus dorsal; the *alæ nasi* dilating considerably with respiration; he was without herpes; the pulse was full, soft, and 92, respirations 46, temperature 102·8. The tongue was moist and thickly furred; the sputum was rather red than rusty, and of quite fluid consistence. Over the base of the right lung, up to about the fifth rib, was tubular breathing, crackling crepitus, and increased voice resonance.

The same evening at 11 P.M. pulse was 84 and respirations 54. The temperature had risen to 104·5, its highest point.

On the following day (fifth of illness) pulse was 88 and dicrotous, the respirations 56, temperature 102·5; a slight herpetic eruption had appeared on the upper lip. In aspect the patient was little changed, the face being still much flushed. The expectoration was watery and blood-stained, the urine had a specific gravity of 1025, was without albumen, and showed abundant chlorides; the tubular breathing remained, with less crepitation. That evening the pulse was 88, respirations 48.

On July 3, the sixth day of illness, he was found perspiring freely, the sputum was more viscid, the morning temperature was 103·2°, the evening perceptibly *lower*, 103·1°. Both by these phenomena, and still more in his altered aspect, was this day marked as the crisis of the fever. On the following day, the temperature continuing to fall, insomuch that the evening temperature was a degree lower than that of the morning, pulse was 90 and respirations 42. On the 5th, or eighth day of illness, redux crepitation was audible



over the base of the affected lung. The pulse had fallen to 64, but the respirations were 44. The temperature was now normal. On the following day, and for the next four, bronchitis was developed; rhonchus became audible over both lungs, the sputum became more abundant, but now colourless. The evening temperature was pretty constant at a little over 101°, the morning a little over 99°. The cough had changed its character, and was more troublesome and constant. On the 10th, however, these symptoms began to subside, and now the resonance of the right lung was restored, there remained no small or medium crepitation, and no trace of tubular respiration. On the 14th the man got up; he was convalescent on the 21st, and on the 26th he went out recovered.

In the course of the fever the man had exceptional wasting, but rapidly recovered flesh so soon as this had subsided. There was no reason to suspect structural lung disease.

This case has some unusual features. There was no rigor; the sputum was never of the characteristic rust colour—at first it was blood, and later a blood-tinged watery secretion, with but little viscidity; the pulse was never frequent, and with a respiration quickening from 46 to 54, it fell from 92 (its highest frequency) to 84; the chlorides were never deficient; bronchitis interrupted recovery.

Yet, with these accidents, it was pneumonia and nothing else. In aspect, temperature, defervescence, physical signs, as well as the mode of recovery on the part of the lung, it might even be called typical. The persistence of the chlorides in the urine along with the unusual character of the sputa, is of interest, in reference to the hypothesis of Dr. Beale which connects hepatisation with the disappearance of these salts.

It is a suggestion of Dr. Morehead ('Diseases of India,' p. 314) that solidification depends sometimes upon 'stagnated blood in the close-set meshes of the pulmonary capillaries.' Upon that hypothesis it may be conceived that in a given instance actual inflammatory exudation would be little or none. In such a case the determination of the chlorides to the lung by means of this discharge from the blood and their elimination by the sputa would not occur, and accordingly the corresponding absence of these salts from the urine would not occur either.

The infrequency of the pulse when there was high pyrexia and notable dyspnœa, though very unusual, is seen from time to time both in pneumonia and continued fever.

As for the relation between the bronchitis and the pneumonia it was accidental. The ward was draughty, the patient's bed exposed, and the wind in the east with a temperature remarkably low for the season. So the man took cold, as any other might. It is especially to be observed that this catching cold *in no way retarded the progress of the lung towards recovery.* The bronchitis arose quite separately, as the pneumonia was departing ; it persisted for about four days, while the lung was undergoing resolution, and the final recovery was not retarded by its occurrence.

To the foregoing narratives of pneumonia, severe and threatening, yet in every instance recovering rapidly so soon as the disease 'took the turn,' may be appended in brief outline, lest the fatality of the disease should be underrated, the circumstances of a case which without much warning took a fatal course, death occurring when a favourable 'critical' change might have been expected.

CASE VII.

Pneumonia fatal within ten days—Highest temperature 103·2—Commencing disease of kidney.

Thomas G., thirty-nine, carman (admitted into Dr. Fincham's ward November 18, 1873), was suspected of intemperate habits, but believed to be in perfect health until six days ago, when he was seized with rigor. He has now a flushed face, with herpes on the lips. Tongue is brown and dry, pulse 130, weak and compressible ; respirations 44, temperature 103·2°. Over nearly the whole of the right lung respiration is tubular, with corresponding percussion dulness and some fine crepitation ; there is no albumen in the urine, and only a trace of chlorides. He is put upon beef-tea, and given ammonia in doses to stimulate, and brandy.

The following day (the seventh from the rigor) the skin is warm and moist, and tongue less dry ; he has had some sleep, the pulse is 140 and less compressible, temperature is 102°, and respirations 40 ; the physical signs as yesterday, save that crepitation extends higher up.

On the 20th his condition was one of great prostration, the pulse somewhat more frequent; other things the same. On the 21st (the ninth day), the temperature being 102·4°, pulse 138, and respirations 46, he exhibited all the signs of approaching death, which arrived some hours after midnight.

Post-mortem Examination, November 22, 1873.—Body well nourished ; mark of blister on chest and abdomen.

The left lung is crepitant throughout, much congested posteriorly, and very œdematous, a large amount of serum escaping on section;

adhesions at base of this lung; no fluid. The bronchial mucous membrane markedly thickened, tough, and fibrous.

The right lung is universally adherent, a small amount of coagulated lymph on pleura; as to the upper third of its upper lobe it is intensely hyperæmic, yet crepitant. Elsewhere this lung is completely solid; its cut surface is granular. The consolidation is grey, and most perfect as to the lower lobe. Between this and the hyperæmic apex mentioned above, is a portion intermediate between hyperæmia and absolute solidity.

The heart normal as to size and thickness of walls, whose texture is very firm; there is much sub-pericardial fat. The left ventricle is firmly contracted. In all the other chambers are firm (post-mortem) clots. All the valves are competent, and orifices of normal size. There is some atheroma at root of aorta; the coronary artery apertures are patent. There is a marked absence of fluid blood. The other organs call for no remark, except the kidneys. Of these the cortical substance of the left is somewhat diminished; its weight 7 oz.; of its fellow 6½ oz. The spleen is very pale.

The case illustrates the value of the pulse as a prognostic sign when the temperature is not very high nor the respirations very frequent. On the sixth day the pulse was 130, with temperature 103·2°; on the day following, with some general improvement, a lower temperature and somewhat reduced rate of breathing, the pulse had risen to 140; it was by this mainly that the man's actual condition was indexed. There was no marked delirium, and the prostration, which became extreme near the time of death, was not great at an earlier period.

The organs, apart from the lungs, were mostly healthy, but there was some atheroma of the aorta, and the cortex of one kidney was diminished. The condition of the bronchial mucous membrane of the unhepatised lung indicated antecedent disease by fibroid increase. The case seemed one which with a healthier subject might probably have recovered.

APPENDIX D.

CLASSIFIED TABLES OF 94 FATAL CASES EXAMINED AFTER DEATH AND ASCRIBED TO PNEUMONIA.

THE cases which follow are very unequal in the amount of their information; they are described in the very language of the original narrators, with abbreviations, but without omission of any matter of importance. From the space the tables would occupy printed *in extenso*, I have been compelled in the present volume to omit Class I. altogether. It relates to hepatisation occurring in the course of lingering diseases, and ascribed to 'low' or 'latent' pneumonia. The diseases it comprehends are very various, but have the common feature that death is long delayed. In many cases the patient is exhausted by some long-continued flux—by diarrhœa, or the constant drain of an abscess. Malignant disease is not uncommon; in one instance the subject died simply starved, owing to schirrous cancer of the œsophagus. It is rare in these cases for any attention to be called to the chest during life. In most of the 45 cases the consolidation is spoken of in language identical with that used in describing pneumonia elsewhere. The lungs are described as 'extensively inflamed,' 'infiltrated with lymph,' &c.

The seat of this consolidation is such as we should expect from its supposed mechanical origin. The posterior and inferior portions of the lungs, that is to say, are most affected, and both participate in the change pretty equally, the solid tissue shading off gradually into that which is merely blood-laden and airless. In 25 (more than half) hepatisation occupies both lungs symmetrically at their lower and depending parts—in that situation, that is, where it is usual to meet with signs of passive engorgement. In 9 only of the whole number was there any certain indication of recent pleurisy. In 1 of these 9 (the only case of the kind out of the 45) pericarditis existed. There are 3 cases out of the 45 where, with the symptoms of continued fever, consolidation is present, not, as with the others, at the lower lobes, but at the apex of one lung or of both.

The remaining classes may speak for themselves. In Class II. hepatisation occurs as a consequence of failure in the function of a secreting organ, or owing to any cause which gives rise to imperfect elimination. This is oftenest met with in connection with diseased kidneys. The illustrations under this heading differ from the latent ones, inasmuch as, for the most part, the implication of the lung is sufficiently apparent during life, and recognised as consecutive on some definite disease elsewhere. The cause of hepatisation in these cases is expressed by saying that the relation between the blood and its channels is disturbed, that the circulation is thence impeded, and that so exudation takes place. In this class general and symmetrical consolidation is exceptional ; the hepatisation resembles nearly that of true pneumonia : it is confined to one lung, its boundaries are sharply defined, pleurisy exists along with it, its chief seat is often in the upper part of the lung. Anatomically it is not always possible to dissever this consecutive disease from simple idiopathic pneumonia. As to its origin we are left to suppose that the irritation of the vitiated blood excites in the lung, now in this part and now in that, a process which, though differing in rate of progress, is, in its histology, strictly analogous to the inflammatory process.

In tabulating 30 supposed examples of this form of disease (Class II.), 8 occurred in which it seemed probable that mechanical obstruction had its share in the result. Excluding such as are of a mixed kind, we have, out of 23 cases, 8 in which the kidneys were markedly granular ; in most of the remainder these organs were enlarged—in some, as it would appear from the surrounding circumstances, owing to 'amyloid' change, in others as a consequence of scarlatina or acute nephritis. In at least 13 of the 23 cases recent pleurisy is present. Of the rest it is usually stated, either that the pleural cavity is obliterated or that old pleural adhesions existed. Six cases are of pneumonia of the upper lobe : in 5, of the right alone ; in 1 only, of both apices. In a seventh case the left lung is hepatised as to its upper two-thirds. In 6 cases the whole of one lung (or very nearly the whole) is hepatised, viz. the right lung in 4, and the left in 2. Pericarditis coexists in 6 cases.

In one of these ((22), an instance of advanced granular degeneration of the kidneys), the consolidation would appear to be due to hypostatic congestion ; in the other 5 *the right* side is hepatised, wholly in 3, and as to its upper lobe in 2.[1]

In Class III. are recorded some instances where hepatisation appears to have depended almost entirely upon obstruction.[2] This form of disease is best illustrated by cases of mitral valvular obstruction. I have recorded 16 such. Excluding 4 of these, where the hepatisation seems to have been somewhat modified by the presence of pericarditis, we have 12 cases left as typical examples of the manner in which hepatisation takes place as the consequence of obstruction. As might be supposed, the two lungs participate equally, or nearly equally, in the change, which is most marked at their lower and depending parts. Often there is some amount of extravasation mixed up with it, and sometimes this 'pulmonary apoplexy,' as it is called, exists most in one lung, and hepatisation most in the other. Yet the hepatisation is in itself of the completest kind, and in some instances, as will be seen, is specially described as due to the infiltration of lymph. The stage of grey hepatisation seems rarely to be reached, and pleurisy is a rare accompaniment. The patients for the most part trace their first failure of health to an acute rheumatic seizure, and will describe former attacks of so-called pneumonia. Other cases in this class seem to have their origin in a feeble action on the part of the heart owing to its fatty degeneration and the impediment of an adherent pericardium. (See Cases 6, 9, 11.)

Coming next to Class IV., 17 cases of simple, uncomplicated pneumonia (three of them double) are all the instances that are to be discovered in searching the records of twenty years at a large metropolitan hospital ; and in this number are included 5 where pericarditis coexists. It is true there are some few cases of a doubtful character to be found in the other tables that ought perhaps to be included here, and so bring the number up to 20, or thereabouts. On the other hand, there are instances amongst these 17 where, although the pathological definition is satisfied, the clinical history forbids us to suppose that the patient had not long been the subject of disease. What first strikes us in glancing at this list is the constant presence of pleurisy along with the consolidation. Next, it is observable that in a majority of these cases either the whole lung is struck, or that it is the upper lobe which is solely or mainly affected. Where this is not so—where the lower portions of the lung are alone solid—we have just those cases which are most equivocal in their history, and most suggestive of some primary disease which has eluded us. In fact, the statement that simple inflammation attacks the lower lobes by preference seems to be, so far as the fatal cases go, the reverse of the truth. Thus in 5 cases the chief seat of the hepatisation is one apex (in 3 the left and in 2 the right apex); and in 6 the whole of one lung is hepatised (the *right* lung in all but one case (5), where the whole of the left lung is inflamed along with the base of the right). Hepatisation is uniform throughout 5 of these 6 cases; in the sixth it is double, occupying the whole of the right lung and the upper part of the left. Omitting 3 of the cases (7, 11, and 15, two admitted moribund, and all without certain history), the average duration of illness in the remaining 14 is about 8½ days. Delirium is so marked a feature in this affection as to have received special notice in these fragmentary reports, in 7 out of the 12 cases of which there is a complete history. In 3 of these (all men) it was of violent character, resembling delirium tremens.

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[1] Vide 7, 11, 16, 17, 18, Class II.

[2] It will be observed that some two or three of the cases of contracted mitral orifice do not exhibit *marked* hepatisation. These are included as helping to illustrate the general effect upon the lungs of this particular kind of obstruction.

The association of *pericarditis* with hepatisation of the *right* lung, to which I have already made passing allusion, is again noticeable in 3 cases out of the 4 where that inflammation was present. In the fourth case the hepatisation is double, though the upper lobe of the *left* lung exhibits the most advanced stage of it.

This connection is so unaccountable that one hesitates to accept it on light grounds. Here is an analysis of all the cases tabulated in which pericarditis and hepatisation coexist. In 23 cases of recent pericarditis it is the *right* lung which suffers in 16, either solely (which is the rule), or with very slight participation on the part of the left lung. Of the remaining 7 cases 4 are not distinctive, that is to say, one exhibits extravasation of the right lung without pneumonia, in 2 the lower parts *of both lungs* are hepatised (the right most in one of them), and in the fourth case the upper lobe of the right lung is hepatised along with all the left. In none of these 4 cases is the pericarditis stated to be recent; in two, at least, it is evidently of old standing. There remain 3 apparent exceptions. Two record hepatisation of the lower lobe of the left lung, together with a thin layer of recent lymph over the heart; the third has honeycombed lymph in the pericardium, along with hepatisation of the back of the left lung. From the situation of the consolidation, and still more from the *absence of pleurisy* in all these three cases, they would appear to depend on hypostatic congestion rather than on true pneumonia.

It may be stated generally, therefore, that wherever in the whole series of cases recent pericarditis is associated with marked pneumonia, it is always the *right* lung which suffers either mainly or solely. In other words, *these Tables do not contain a single case of extensive hepatisation of the left lung only along with recent pericarditis, while they contain at least* 10 *in which that is the condition with respect to the right lung alone.*

I have placed by themselves (under Class V.) 20 cases of lung-consolidation, where death was ascribed by the narrators to 'low' or 'latent pneumonia.' Only 5 of these seem due to hypostatic congestion; the remainder cannot be accounted for in any mechanical way. Thus there are 10 cases where the upper lobe is the seat of consolidation; and it happens, strangely enough, that in all these it is the *right* lung which is so affected. Of the remainder, the whole of one lung is affected in 5, but in all save 1 of these the disease is most advanced at the apex. Recent pleurisy existed in 5 cases only out of the 20, though the value of this sign is greatly diminished from the pleura being in several instances obliterated by old adhesions. Recent pericarditis was found in only one case in association with consolidation of the upper portion of the right lung, and dependent apparently on granular degeneration of the kidneys.

Consolidation of the lung is said to occur in a similar latent manner in connection with delirium tremens, and indeed two such cases appear in the table I am speaking of. Dr. Stokes alludes to this as one of the forms of 'typhoid pneumonia.' 'The disease,' he says, 'commonly attacks the left lung, particularly in its lower portion, and is constantly overlooked.' It appears, on the contrary, that in the 5 cases answering to Dr. Stokes's description, to be found in all my Tables, both lower lobes were affected in 2, while the remaining 3 are marked cases of hepatisation of the upper lobe of the *right* side—2 of them having also pericarditis. (See Chap. vi., p. 89).

Class VI. contains eight cases, which I have preferred placing apart, owing to particular circumstances which in each instance render classification uncertain.

Some other examples, indeed, offered difficulties in this respect, and may perhaps be considered as wrongly placed. For instance, Cases 8 and 11 of Class II. may belong most to Class IV., while (as is mentioned at the place) 15, 16, and 17 of the same class have features in common with Class III. On the other hand Cases 11 and 13 of Class IV. might properly from their histories be placed under Class II. I have throughout maintained that a rigid classification is impossible, and this blending is but an illustration of that text. It is to be considered, besides, that the brevity of the records may give rise to uncertainty which would disappear if fuller information were attainable. The cases themselves are by many hands, and, as is explained in Chapter II., their arrangement is, in some measure, that of the narrators. I would ask the reader, therefore, to take these ninety-four examples as they come, and on their own merits. They afford a body of evidence unmanipulated for or against the views set forth in the preceding treatise.

CLASS I.

Table of Cases of Hepatisation occurring in the course of lingering Diseases, and ascribed to low or latent Pneumonia.

(Omitted for want of space.)

(*Vide ante, p.* 269.)

CLASS II.

Table of Cases of Hepatisation the result of Blood-poisoning.

Reference to Post-mortem and Case-books.	Nature of case, &c.	Post-mortem appearances: site and extent of hepatisation.	Co-existence of pleurisy, or of pericarditis.
XLIX. 53 M. 63. 1.	Rapid sinking after simple fracture of tibia and fibula. Of early history it is stated that for years he had been 'a martyr' to gout. 'No symptoms presented themselves to call attention to chest' (Surgical Report).	Left lung congested posteriorly; lower lobe of right lung much consolidated 'from effusion of lymph and serum in its parenchyma,' of a light dirty brown. Kidneys small, rough, and granular.	Some recent lymph on surface of hepatised lung. Pericardium not mentioned.
LII. 236. M. 57. 2.	Admitted at first under surgeons for 'cachectic rupia' of six weeks' standing. Two days after, he was observed to be in a very depressed state, with cough and oppression of breathing; was stimulated and blistered. The following day he was in a typhoid state, with dry tongue and difficult articulation, but no confusion of mind; lying constantly on his left side. He was cupped to ℥x., and afterwards dry-cupped. He sank the same day. Three days in hospital.	The lungs on both sides full of reddish frothy fluid; but all parts floated, except the middle lobe of the right lung, which was almost entirely consolidated and of a light buff colour—the grey hepatisation. Bronchial tubes very vascular and full of thick mucus. Some old inactive cretaceous deposit at both apices. Kidneys weighed 8 oz., were highly granular, and with diminished cortical part and numerous cysts. The heart weighed 15 oz.	A little reddish fluid in both pleuræ, and some old adhesions in one; no lymph. Pericardium not mentioned.
L. 55. M. 44. 3.	Admitted with pain in the chest, cough, and dyspnœa. Had had dropsy of legs and face at beginning of illness four months before. Cough had come on during last	Upper lobe of right lung consolidated by recent inflammation; of greyish mottled appearance; the middle lobe red and condensed; the lowest lobe congested only.	Lymph an turbid fluid in left pleura, so that left lung

	fortnight with such great debility that he had kept his bed for that time. Treated by bleeding and antimony. Two days in hospital.	Heart hypertrophied; valves healthy. Kidneys rather large, pale, mottled.	was compressed thereby, its lower lobe being impervious to air.
XLVII. 219 F. 67. 4.	Wretched cachectic person. Admitted for general pains about limbs; severe pain in epigastrium; frequent vomiting. The last symptom more especially for nine months; pyrosis, &c.; no cough. Died by gradual sinking; vomiting being chief symptom. Six days in hospital.	Upper lobe left gorged with serum, but crepitant; lower lobe throughout quite solid; in parts with red hepatisation, and in parts with grey hepatisation, readily breaking down. Lower lobe right lung inflamed; larger portion in red hepatisation. Upper lobes crepitant, loaded with serum. A small tubercle at one apex. Kidneys much diminished; cortex nearly all absorbed.	Old adhesions left side; layer of recent lymph on lower lobe. Firm adhesions right side; recent lymph at lower part.
LIV. 123. M. 28. 5.	First admission in March 1853 with severe bronchitis. He had been subject to cough and dyspnœa for five years. His face was dusky, and once the sputa were streaked with blood. He left the hospital in April, still with cough and rather dusky face. Re-admitted in May 1854, in a more depressed state. Had not been free from cough in the interval; last taken suddenly worse two days previous, with pain in left side. Sputa were abundant, frothy, and muco-purulent. No mention of side-pain after admission. Pulse was weak and rather frequent. Stethoscopic sounds those of bronchitis, except some amount of dulness. Five days in hospital.	Body in good state. Lower part of right lung was emphysematous. Almost all remaining portion in a state of hepatisation, solid, and in places yielding a greyish fluid. Upper and middle parts presented a granular surface on section. At one part of middle portion of upper lobe a small amount of fluid within an irregular cavity. Left lung emphysematous; at lower part congested, crepitant. The bronchial tubes vascular, and containing much mucus.	In right chest recent adhesions, some fibrin, and yellow fluid. Heart and pericardium natural. Blood thin and watery.

Reference to Post-mortem and Case-books.	Nature of case, &c.	Post-mortem appearances: site and extent of hepatisation.	Co-existence of pleurisy, or of pericarditis.
IX. 238. M. 31. 6. -	An intemperate sailor. Four or five months before admission he walked thirty miles in the rain. He then sat down in a public-house and let his clothes dry on him while he drank. The next morning œdema of legs appeared, and dropsy continued up to time of admission. Urine was then highly albuminous. After a few days, some blood-tinged sputum was observed. Progress of the case marked by orthopnœa and increased anasarca, death occurring, after long lingering, three months and a half after admission.	General dropsy. The upper lobe of the right lung is hepatised and condensed from infiltration of lymph. Upper and part of lower lobe of left lung is in a similar state. The kidneys are small, smooth, and mottled; their cortex not diminished.	Fluid in left pleura and old adhesions of right.
LIII. 73. M. 48. 7.	A fat, flabby, ill-conditioned man, evidently in the habit of free drinking. Admitted for rheumatic pain in hands and shoulders of four days' duration. The hands were slightly swollen. He had great difficulty in moving; was bled twice (to ℥xviij. in all). The sweat had the usual acid odour (he had had cough for three months). At each apex sonorous râles were heard with the expiration; but there was no dulness. On the fourth day, tremor and tendency to delirium. Both these increased on the fifth day, and the sounds of pericarditis were heard. The wandering was now very similar to ordinary delirium tremens. The patient now gradually sank, and died on the twelfth day.	Left lung somewhat congested posteriorly; but crepitant throughout. The lining membrane of the bronchial tubes was inflamed and loaded with frothy muco-purulent fluid. The *upper lobe* of the right lung was completely solidified from red hepatisation. The tissue of the organ was soft, and easily broke down. The heart was much enlarged, weighing 23 oz.; all its valves were healthy. The kidneys were large, weighing 15 oz.; healthy in structure.	Recent adhesions between pleuræ, corresponding to inner side of upper lobe of right lung and surface of pericardium. Three ounces of serous fluid in the pericardium; a shaggy layer of recent lymph on its visceral surface.

F. 28. 8.	days ago took cold. Had rigor, cough, and pain in *left* side; was leeched and blistered. On admission was very prostrate, fainting when raised; loud crepitation was heard at the lower part of the left side. Second day rubbing is heard at the base of the heart; the sputa were yellow, copious, muco-purulent. (Treatment, calomel and opium.) Loud gurgling was heard all over the chest. She died on the third day from admission.	Left lung much compressed; numerous very old adhesions on the right side. All the right lung hepatised and easily breaking down. Other organs not mentioned.	lowish fluid in pericardium. The serous membrane covering right auricle thick and opaque. Outer surface of left lung covered with thick layer of recent lymph. Pleural cavity containing large quantity of straw-coloured fluid.
LX. 136. M. 64. 9.	A hard drinker. Ill for a month with boils or superficial abscesses about nates. A week ago, when getting out of bed, slipped and broke his ribs. Three days before admission, dyspnœa. On admission orthopnœa and severe dyspnœa (dulness and large crepitus over upper lobe of right lung). Ordered squills, bark, and ammonia. On second day, calomel and opium and wine. On third day was restless and wandering; breathing more difficult, much tenacious matter sticking about the mouth. Died next morning. Three days and a half in hospital.	Body rather fat. Eighth and ninth ribs of right side fractured, with no attempt at union. Upper lobe of right lung throughout in a state of grey hepatisation (advanced); the lower lobe spumous. Heart healthy; extremely full of decolourised clots on both sides. Spleen diffluent; kidneys slightly granular; the pelvis containing much fat.	Pleuræ both adherent; not injured by the fracture.

Reference to Post-mortem and Case-books.	Nature of case, &c.	Post-mortem appearances: site and extent of hepatisation.	Co-existence of pleurisy or of pericarditis.
LV. 97. F. 59. 10.	A debilitated-looking woman. A month ago she was attending a neighbour in childbirth, and there caught cold. Two weeks before admission the cold became worse; cough, with slight spitting, succeeded, and she had shivering and pain in the loins. On admission she had pain in left side, and cough (her general condition is not further described); pulse was 106. She was blistered, and given calomel and opium. After six days she was free from pain, and the drugs were stopped. Next she was observed to be feeble and anxious, with rapid pulse and dry tongue; sordes on lips, &c. (twelfth day). Fourteen days in hospital.	Emaciated. The lower lobe of left lung and the back part of its upper lobe in a state of grey hepatisation, solid, sinking in water; the parenchyma infiltrated with lymph and pus; the front of upper lobe healthy. Lower lobe of right lung like lower lobe of left; other parts of both lungs œdematous. Heart healthy. Other viscera healthy (not specially mentioned).	Old adhesions both sides, but much more extensive on the right. (Pericardium not mentioned.
LV. 205. M. 26. 11.	A labourer, who had been six days ill 'with cold and cough,' and had spit a very little blood. On admission was flushed, and with full bounding pulse; not rational enough to give his full history; same night he became very noisy; the urine (suppressed for a time) was found to be albuminous; head very hot, great thirst; some crepitation and dulness at lower lobe of right lung. By the third day the physical signs and febrile symptoms had increased; fourth day twitchings of hands and low muttering; tongue now (fourth day) became black and fissured, with sordes on lips and congested face; fifth day some wine was given (an-	Body in good state. No œdema. Whole of right lung completely hepatised (grey); several small abscesses in various parts. The left lung was natural. The bronchial tubes are not mentioned. Kidneys were large and solid, with very yellow cortex, greatly mottled on the surface.	Right pleura obliterated; a little fluid (yellow) in left. Pericardium coated with thick layer of recent lymph.

	continued). The expectoration was now brownish, it had before been rust-coloured. Pulse became less frequent and tongue cleaner. Remained two days in this favourable state, and omitted all medicines. On the eleventh day began to sink, and had constant vomiting. Eleven days in hospital.		
LV. 207. M. 42. 12.	An intemperate man. Seized five days before admission with pain in left side and vomiting. On admission, urine rather albuminous; tongue dry and dirty; full quick pulse. Vomited whenever he moved. Sputum frothy, muco-purulent (cough and some expectoration for years). Given stomachics, &c. Retching returned. He sank slowly. Six days in hospital.	Whole of left lung (except upper lobe, which was natural) firm and full, in state of grey hepatisation. Lining of bronchial tubes of affected part very highly vascular. The same tubes in upper lobe comparatively pale. Right lung quite natural. Kidneys cysted, granular, with adherent capsules. Liver pale. Other organs natural.	Old and recent adhesions left side, and much sero-purulent fluid. Heart and pericardium natural.
LXII. 43. M. 28. 13.	A labourer, admitted with febrile symptoms and weak pulse; pain under right nipple, and slight difficulty of breathing; spitting rusty mucus. Had been ill five days. The defined symptoms above only two days. No dulness detected till second day. From the physical signs, consolidation appears to have begun at the upper part of the lung. Given antimony wine; and later, calomel and opium every four hours. On the fifth day became very violent, and continued noisy that day and the following; vomiting after everything, and obstinately refused food, &c. No great dyspnœa now, and the spitting had almost ceased. Sixth day, became cold and collapsed, and died. Six days in-patient.	Right lung completely solid throughout from red hepatisation; in a few places (chiefly in the upper lobe) broken down. Left lung healthy. Both kidneys very granular on their surfaces; of large size; the capsules thickened. Heart not mentioned. Liver large. Other organs healthy.	Right lung surrounded by old adhesions. Left surrounded by adhesions (old?).

Reference to Post-mortem and Case-books.	Nature of case, &c.	Post-mortem appearances: site and extent of hepatisation.	Co-existence of pleurisy, or of pericarditis.
LVI. 80. M. 13. 14.	Had scarlatina a month before, which left slight œdema. Four days ago he had a fit, and a second at time of admission. On admission was blooded (amount not stated); blood highly buffed and cupped; became wildly delirious; urine was very albuminous. He lay wholly on the right side. Two days or less in hospital.	Right lung very congested. The lower lobe in a state of extreme red and grey hepatisation, very few vessels or tubes being visible. Kidneys pale; stellate on surfaces; very opaque; a yellow cortex. Other viscera natural.	Reddish fluid in both pleuræ; in right sac some recent soft fibrin.
LXII. 25. F. 21. 15.[1]	Admitted fourth day of acute rheumatism, having had two previous attacks. Bled to ℥xij.; cal. gr. iij. and opium gr. j. every night; alkalies. Third day very pale and white, with pain in right side and cough. Fourth day, blistering and antimony wine. By the eighth day cough was much better; she was then up, and had been so two or three days. Twelfth day, ulcerated throat observed; this getting worse up to twenty-second day, when pain in right side and dyspnœa occurred; the sputa becoming blood-stained; the urine full of albumen; stridulous breathing, as from affected larynx. Died two days after the acute symptoms.	Right lung hepatised throughout; light buff, mottled with streaks of reddish-brown; infiltrated with cells like pus, except that acetic acid displayed numerous fine granules instead of the characteristic compound nucleus. All the air-cells of a thin section were completely filled by the exudation; the walls not thickened. Ulcers on tonsil, and a 'diphtheritic' membrane on pharynx; soft palate and larynx. Left lung healthy. Recent lymph about mitral and aortic valves, the former much thickened and inflexible. Right kidney with dilated pelvis and shrunken cortex; no obstruction being discovered. Other organs healthy.	Much serous fluid in pericardium, and some recent lymph. Right pleura obliterated; much recent lymph at its lower part. A few old adhesions of left pleura.
LXII. 60.	Ill one year and nine months; illness beginning with 'rheumatic gout,' followed by	Slight œdema of the body. The whole of the right lung red, solid, and gra-	Tightly adherent and

16.	dyspnœa. Took antimony and calomel. Twenty-two days.	conspicuous from their white colour, and arose apparently from emphysema of some lobules. Some small amount of hepatisation, lower lobe of left lung. Heart pale, its cavities dilated; recent fibrin on its valves. Heart and pericardium weighed 20 ounces.	of deposition, of which the inner was the more recent.
LXII. 107. M. 18. 17.[1]	Acute rheumatism with heart affection at the age of 11. Present attack began with wandering pains, two weeks ago. Ten days ago had pain in the præcordial region, which remained up to time of admission. Admitted gasping for breath, and with orthopnœa; pulse 108; much tenderness over the heart and left chest generally. Treated by alkalies and calomel and opium. Some amendment took place, and the pulse sank to 94. Fifteenth day, some œdema of the legs is noticed. From this time he varied from day to day as to dyspnœa, &c., the swelling increasing. He died exhausted. Forty-six days.	Legs œdematous. The aortic valves thickened with old fibrous deposit; in one of them was a small hole; an abrupt ridge of deposit of several dates on the inner surface of the mitral valve. The right valves were healthy, and the left lung. The right lung slightly solidified, as if in the earliest stage of pneumonia. Heart weighed 1 lb. 15¼ oz. Cavities dilated. The kidneys were increased in size.	Pericardium greatly thickened, and adherent to the whole surface of the heart. (Pleuræ not mentioned.)
LXIII. 132. F. 16. 18.	Chorea; violent convulsions, dying soon after admission as if falling asleep. History of rheumatism. Some hours in hospital.	Middle and upper lobe of right lung in a state of incomplete hepatisation ('the first stage'). The spleen dotted with small white specks smooth to the touch. Recent lymph on valves of left side of heart.	Old lymph on pericardium.

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[1] Cases 15, 16, and 17 have much in common with Class III.

Reference to Post-mortem and Case-books.	Nature of case, &c.	Post-mortem appearances: site and extent of hepatisation.	Co-existence of pleurisy, or of pericarditis.
LXIII. 137. M. 50. 19.	Good health till two months ago, then œdema and cough, the urine becoming scanty and bloody. Admitted wheezing, with râles audible, and with bloody urine. Diuretics given. Œdema subsides in four days. On the seventh day sputum becomes blood-tinged; pulse rose; pain in right chest. He became very livid, and died. Eleven days in hospital.	Whole of left lung consolidated, and apparently swollen with grey hepatisation, except the apex. The bronchial tubes healthy. Kidneys smooth, large, $16\frac{1}{2}$ oz. in weight; ecchymoses throughout their cortex, and much congestion. Large heart (23 oz.); its left ventricle much thickened; soft atheroma in the aortic valves.	Much turbid fluid in left pleura, and thick recent lymph.
LXIV. 43. M. 2. 20.	Admitted moribund. Scarlatina a month before, followed, it was thought, by recovery. Two weeks ago chronic twitching. Day before admission dyspnœa and croupous breathing. A few hours in hospital.	Lower three-fourths of right lung solid, so as to sink; it had a dark brownish colour, mottled with light brown spots like circumscribed grey hepatisation. Lower lobe of left lung contained a few similar spots. A little recent lymph on inner surface of mitral valve; small speck of the same on the aortic valve. Kidneys very pale, and mottled with irregular vascularity; the cortex increased; weight $4\frac{1}{2}$ oz.	Recent lymph in right pleura, and a little serous fluid. Left pleura natural.
LXIV. 175. M. 28. 21.	Ill a month. Debility, œdema, pain in the chest. Two weeks' cough; thick uncoloured mucus and some specks of blood; respiration something hurried. Given squills, digitalis, and acetate of potash. Death quite sudden and unexpected. One	All upper lobe of left lung and greater part of lower lobe firm, red, and granular; the tissue friable and heavy; most parts sank. Right lung full of serous fluid. Kidneys enlarged (weight 20 oz.); smooth, vascular, a few stellate veins on surface. The	Pleuræ not mentioned (natural?).

		paler than natural, owing to the presence of some opaque whitish matter. Heart natural, except for one or two beads of recent lymph on the mitral valve.	
LXIV. 259. F. 45. 22.	Admitted in a state like fever. Only partially conscious. No history could be obtained. Lived in hospital some hours only.	Lower lobe of left lung and greater part of upper in condition of diffuse hepatisation, exuding purulent fluid in many places, the consolidation not generally complete. Lower lobe of right lung in state of red hepatisation. Pericardium completely filled with recent lymph, layers thereof in adhesion to each other. Left ventricle hypertrophied, valves natural. Kidneys finely granular, shrunken, and with diminished cortex. The organs chiefly consisting of fibrous tissue.	Some flakes of recent lymph on surface of left lung.
XIII. 291. F. 30. 23.	A large flabby woman, ill since her confinement two months ago, much hæmorrhage following delivery; sore-throat later; dropsy set in two weeks back; kept bed for last ten days; winter-cough for many years, and severe dyspnœa. Admitted with extreme dyspnœa and orthopnœa; sputum yellow, and finally rusty; delirium. One day and a half in hospital.	Upper lobe of right lung in a state of grey hepatisation. In several places small circumscribed deposits of pus, which might have been regarded as minute confluent vomicæ, had any tubercle been discovered. The whole lobe solid and livid. The lower lobe and left lung natural.	Sero-purulent fluid in right pleura, and a thick layer of recent shaggy lymph.

CLASS III.

Table of Cases of Hepatisation from Obstruction.

Reference to Post-mortem and Case-books.	Nature of case, &c.	Post-mortem appearances : site and extent of hepatisation.	Co-existence of pleurisy, or of pericarditis.
LII. 95. M. 47. 1.	Out of sorts for three weeks. Supposed healthy before. Some years ago had rheumatic fever (marks of leech-bites over heart). Symptoms : cough, dyspnœa, and anasarca. Heart's action quick and difficult to appreciate. Urine very albuminous ; excessive dyspnœa. Rather rapid death. Two days in hospital.	*Exceeding contraction of mitral orifice* and dilatation of left auricle. Weight of heart $8\frac{1}{4}$ oz. Lower lobe of left lung hepatised ; not so the right ; both congested. Slightly granular kidneys.	Old adhesions in right pleura ; in left, both old and recent adhesions. A little fluid only in pericardium.
LI. 89. F. 16. 2.	The subject of frequent rheumatic attacks. Admitted for palpitation ; afterwards some dropsy appeared. There was a bluish flush on cheek ; a cardiac bruit, and oppressed breathing (no further lung-symptoms mentioned). Rather gradual sinking. Fifteen days in hospital.	Auricles enormously distended by semi-clotted blood ; vegetations on mitral valve : *mitral orifice contracted.* Other orifices and valves natural. Lungs were friable and sank in water ; dark red fluid could be expressed from them. The left was congested merely.	A little clear yellow fluid, and some very firm adhesions of pericardium. Pleuræ very vascular ; in right sac 5 oz. of reddish fluid.
LVI. 72.	Looking 12 or 13 only ; had never menstruated. 'Biliousness' for some time.	Back part of right lung œdematous and containing a brown circumscribed patch the size of a	Fluid in both pleuræ, in

	some blood; got rapidly worse. Eleven days in hospital.	*Mitral orifice contracted to size of end of little finger;* tricuspid also contracted; some hypertrophy of walls.	universally adherent.
LVII. 22. M. 26. 4.	Subject to palpitation and dyspnœa since an attack of acute rheumatism seven years before. Ill with those symptoms six or seven days on his admission; supposed from getting wet. Nine days in hospital.	Lungs in places emphysematous; at lower parts consolidated; firmer and darker patches here and there, as from old inflammation or old-standing extravasation of blood; but in no place was any recently extravasated blood. Liver large and nutmeggy. Heart very large (27 oz.). *Mitral orifice narrowed* by calcareous matter and thickening of flaps; two fingers could not enter. Left auricle very large.	Pleuræ not mentioned.
LVIII. 20. M. 37. 5.	Dyspnœa only; no pneumonia mentioned or treated; intemperate; congested face; frothy sputa. Ill five weeks in a similar manner before he came under treatment. Nearly one month in hospital.	Red hepatisation of upper lobe of left lung; emphysema of the lower lobe. Right lung congested. Bronchial tubes dilated. Hypertrophied heart (20 oz.).	
LIX. 79. F. 17. 6.	Account of an illness (ill-defined) with delirium and increased action of heart, three years before (rheumatism?); has had palpitation ever since. On admission, rheumatic pains about the shoulders; a loud systolic murmur; pulse regular, not very unnatural; no œdema; constant orthopnœa, but no obvious dyspnœa. Given bark and wine. Latterly, œdema and emaciation; mental wandering. Lingered long. Died as fainting. Forty days in patient.	The lower part of each lung in a state of red hepatisation; the inflamed part melting down gradually into the healthy. The pericardium firmly adherent. Heart much hypertrophied; its muscular fibres in advanced stage of fatty degeneration. Some deposit of lymph on aortic valves. Other organs healthy.	Adherent pericardium.

Reference to Post-mortem and Case-books.	Nature of case, &c.	Post-mortem appearances : site and extent of hepatisation.	Co-existence of pleurisy, or of pericarditis.
LVII. 225. F. 40. 7.	For twelve weeks dyspnœa and pain between shoulders. For a month has had œdema of the feet. Urine albuminous ; heart 150 in the minute ; pulseless ; much anasarca, and a livid face. Three days in hospital.	Lungs generally pale and bloodless ; but two patches of pulmonary apoplexy in the substance of the right lung near together, each about a cubic inch. Kidneys rather granular. Some hypertrophy of left ventricle. *Mitral orifice exceedingly contracted*, admitting only point of forefinger.	Adhesions of right pleura.
LVIII. 148 M. 22. 8.	Palpitation and diarrhœa (the former ascribed to overwalking three weeks before) ; in bed for two weeks. No history of rheumatism. Breathing very distressed ; urine albuminous ; anxious to be left alone. Died suddenly, after apparent improvement. Six days in-patient. (Imperfect account.)	Good condition. Heart very large (21 oz.) ; ventricles very capacious ; wall of left much hypertrophied ; wall of right rigid and leathery ; both filled with gelatinous clot. Lower lobes of both lungs in state of red hepatisation. Large fibrinous vegetations on aortic valves, which were small, rigid, and insufficient for closure. *Mitral orifice hardly admitting a finger* ; its flaps rigid ; the ventricular surface covered with fibrinous vegetations. Spleen and kidneys containing large blocks of fibrin.	Old pleural adhesions on right side.
LVI. 67. Lad—age not given.	Admitted dying. History states that he had been ill eighteen months ; much worse for the last six months. An attack of acute rheumatism (date not stated) had left him very anasarcous. Died shortly after ad-	Considerable œdema of lower extremities. Red hepatisation of lower part of upper lobe and of the whole of lower lobe of the right lung. Lower lobes on both sides contained several large, firm, solid, circumscribed patches, where,	A good deal of bloody fluid in left pleura, and little in right.

		Heart of enormous size and universally adherent to pericardium. Liver and spleen congested.	risy right side. Adherent pericardium.
LVII. 276 F. 27. 10.	Dyspnœa for several years. Admitted with that and general œdema; the jugulars being greatly distended. Ten days in.	'Apoplexy' in left lung. The right lung grey and quite solid. The right auricle and its appendix very much dilated; orifice of tricuspid very large. Heart weighed 16 oz.	Fluid in right pleura.
LX. 26. M. 62. 11.	History of acute rheumatism twice, and now for seven weeks. Cough and dyspnœa, with dropsy. Urine is albuminous; sputum rusty, 'pneumonic;' delirium at night; pulse 54, very irregular. Antimonial wine, and later, stimulants. Death by asthenia without dyspnœa. Five days in.	Lower lobe of right lung hepatised. Left lung healthy, but for a patch of apoplexy the size of a nut. Heart large; its cavities much dilated; the walls hypertrophied; decolourised clot in right cavities. The left valves rigid and atheromatous; extreme atheroma of root of aorta. Remaining organs natural.	Serum in right pleura, and a very general lining of lymph. Serum, but no lymph, in the left pleura.
LX. 144. M. 22. 12.	Cab-driver; had had rheumatic fever four years ago, and ever since palpitation. A cough came on, 'from a cold,' three weeks before admission, with spitting and increased palpitation. Urine very scanty and dark; œdema shortly supervened. On the day of admission sputum was blood-stained. He was very sallow and œdematous, and had orthopnœa; irregular heart and pulse (82). Given squills and saline diuretics; twice dry-cupped. The sixth day dropsy increased, he became delirious and violent, and pulse became frequent. Ten days in.	Lower extremities very œdematous. Heart very large, chiefly from distension of its cavities, the walls being weak and thin. The *orifice of mitral diminished* and perfectly rigid from large mass of calcareous matter on both flaps. Aortic valves healthy. Left lung much congested. Whole of lower lobe of right lung in a state of red hepatisation. At its lowest part, amongst the inflamed tissue, a small spot of pulmonary apoplexy. Liver soft and pale. Spleen contained block of fibrin.	Much fluid in right pleura; left pleura adherent. Bloody fluid in pericardium.

Reference to Post-mortem and Case-books.	Nature of case, &c.	Post-mortem appearances : site and extent of hepatisation.	Co-existence of pleurisy, or of pericarditis.
LX. 225. M. 17. 13.	Ill with rheumatic pains for four days previously. For years subject to cough and slight hæmoptysis. On admission, rather acute rheumatism, with tumultuous heart-sounds. No pain in chest ; a very little viscid light-coloured spitting. Bled to ℥xij. ; given calomel and opium ; also ten leeches over the heart (physical symptoms being those of pneumonia). Continued to get weaker and more short-breathed. Always on his back. Sank rather rapidly. About six days in-patient.	Rather large heart ; decolourised clot in all its cavities. The mitral rigid ; *its orifice barely admitting point of forefinger.* Whole of lower and part of upper lobe of left lung in a state of red hepatisation. Back of right lung in similar state, and much loaded with serum. Other organs healthy.	Much clear serum in pericardium. Old adhesions right pleura: left pleura healthy.
LXI. 171. M. 50. 14.	Case nearly illegible. Dyspnœa and cough, sonorous râles being audible in the right chest. Had been in previously with supposed effusion into right pleural cavity. Anasarca came on, and the legs were punctured when sloughing commenced. He lingered four months and a half.	A deep and extensive ulcer on the right leg. A few miliary tubercles at apex of left lung, and some pulmonary apoplexy at its lower part. Right lung entirely consolidated by means of a grey granular deposit, which infiltrated its entire substance. Heart much hypertrophied. Both auriculo-ventricular valves closely embraced by strings of yellow fibrinous coagula, *the openings both somewhat contracted.* Kidneys enlarged, and a little rough.	Fluid in lower part of right pleura. No recent pleurisy. Pericardium adherent by stratified lymph.
LXI. 305. F. 40. 15.	Admitted with dropsy. Sputa generally bloody. Account mostly illegible. One month in-patient.	Some parts of the lower lobe of the left lung were hepatised; some cretaceous masses at the apices. Heart of natural size ; *extreme contraction of the mitral orifice*, which barely admitted the end of the little finger.	

III. 17. F. 50. 5.	Urgent dyspnœa and cough for years; present attack three weeks. Admitted blue and gasping; coarse crepitation at base of right lung, &c. Given diuretics. Forty-four days.	Lower lobe of right lung (the greater part) hepatised; the bronchial tubes congested. Walls of heart thickened and yellow, fatty; *mitral orifice narrowed*, admitted but one finger. Heart weighed 16 oz. Old lymph on the visceral pericardium. Kidneys slightly granular; some depressions and cicatrices on their surface.	Recent lymph in pleura corresponding to hepatisation.
XIII. 164. F. 28. 7.	Dyspnœa and cough for six months (after a cold); never had rheumatism; latterly some dropsy has appeared. Admitted very short of breath, with irregular pulse and loud systolic bruit. Four days after admission the sputa became bloody, but not for long; pulse was always full and sharp, and generally above 100. Cupped and dry-cupped; antimony given and digitalis. Forty-six days in.	Belly distended and fluctuating. Whole of lower lobe of right lung solid, of buff colour, mingled with small patches of extravasation. The left lung, in the same situation, exhibited to a smaller extent the same change. The bronchial mucous membrane was thickened. Kidneys were increased in size; buff-coloured and mottled; the cortex increased in thickness; their weight 18½ oz. *Mitral orifice considerably contracted* and thickened by means of calcareous deposit—only the little finger could be passed; ventricles uncontracted. Some recent adhesions about intestines.	Fluid in left pleura. Heart covered with false membrane of some standing.
IV. 335. 10.	No case. About a day and a half.	*Mitral valve thickened and narrowed*; beads of recent lymph along its auricular aspect, also on the aortic valves. Both lungs largely occupied by red hepatisation irregularly diffused, natural tissue being interposed; left apex natural, right solid. Heart hypertrophied (9 oz.).	Much old lymph in pericardium.

Reference to Post-mortem and Case-books.	Nature of case, &c.	Post-mortem appearances: site and extent of hepatisation.	Co-existence of pleurisy, or of pericarditis.
LXIV. 101. M. 36. 19.	Caught cold, as he supposed, six weeks before from exposure to wet; health previously good but for frequent attacks of rheumatism; rheumatic fever three years ago. Admitted with œdema; ascites and blood-stained sputum; mitral bruit; action of heart regular, and not excessive; highly albuminous urine. Diuretics and elaterium given. Sputum abundant and blood-stained. Day before death erysipelas on left side of nose and some delirium. Fourteen days in.	Slight jaundice; legs œdematous. Greater part of upper lobe of right lung consolidated with hepatisation; the tissue soft and friable, and in some places gray, yielding much sero-purulent fluid on pressure. Small portions of the lower lobe of the left lung friable; bronchial tubes red, not containing fluid. Left ventricle contracted and hypertrophied. Heart weighed 25 oz. Aortic valves very atheromatous, so that they must have been permanently open; around the mitral was an enormous mass of stony deposit, which *narrowed the orifice so as only to admit the little finger*, and probably prevented its complete closure. Liver nutmeggy. Kidneys natural; in places a little yellow. Spleen large and firm.	Old adhesions and half a pint of fluid in each pleura. Pericardium uniformly adherent by old adhesions.
LXIV. 189. M. 13. 20.	Anæmic and wasted; orthopnœa and slight œdema. Three months ill from dyspnœa and chest-pain. Admitted with pulse 120, and much heart-impulse. Ninth day pericardial friction audible, œdema increased, and pains occurred in the limbs (rheumatic). Rather gradual sinking. Thirty-six days in.	Left lung infiltrated with serous fluid. A considerable part of lower lobe of right lung in a state of red hepatisation, sinking in water. Heart hypertrophied; mitral valve thick and stiff from fibroid growth in its substance. Liver congested.	Left lung surrounded by old adhesions. Pericardium universally adherent by a succession of alternate layers of black coagulum and recent lymph alternately (about two of each).

LXIV. 167. F. 19. 21.	Admitted with acute rheumatism, not very severe. The resident apothecary was called to her on the second day. He found her breathing with much difficulty, in a shallow, catching manner, and complaining of pain in the left side, with increase of the rheumatic symptoms. No friction-sound could be heard. Six leeches applied to left side; calomel and opium given every four hours. Next day she was tender, cautious of slight movement, and very breathless; pulse 104. Nothing was made out by auscultation of the chest anteriorly. Heart-sounds were quite clear. Painful breathing, restlessness, and general distress continued till death, the mind remaining clear. Four days in hospital (or two from the seizure of dyspnœa).	Very fat. A small shred of decolourised lymph at beginning of the right middle cerebral artery, not closing the channel; some of the small arteries were irregularly distended with fluid blood. The lower lobe of the left lung was much solidified from pneumonia, and sank in water. The right lung was natural. Along the inner edge of the mitral was a layer of soft recent lymph, of which a few beads were seen on the aortic valves. The pulmonary artery, from its origin to its third and fourth divisions, was filled with coagulum, as was the right ventricle; in many parts of the artery the coagulum was opaque, granular and friable, evidently of some standing; in some places it was elastic and semi-transparent, and was mixed with ordinary black clot; the coats of the artery were quite natural, the clot but slightly adherent. The right ventricle contained a large quantity of yellow elastic fibrin, continuous with the clot found in the pulmonary artery. Some patches of fatty degeneration detected in some of the columnæ carneæ.	Both pleuræ contained a little fluid. Some recent lymph on right lung. The heart uniformly covered with a thin layer of recent lymph.
LXIV. 343. F. 39. 22.	Admitted prostrate, with frequent pulse, dry tongue, &c. Lived some hours only.	Both lungs irregularly mottled, with little red patches approaching hepatisation; here and there patches of complete hepatisation. Both lungs emphysematous. Spleen contained some fibrinous blocks—some hard and recent, others old and soft. *Old thickening and narrowing of mitral*, which was covered with a quantity of shaggy recent lymph.	Pleura not mentioned.

Reference to Post-mortem and Case-books.	Nature of case, &c.	Post-mortem appearances: site and extent of hepatisation.	Co-existence of pleurisy, or of pericarditis.
LXIV. 93. F. 11. 23.	Anæmic; had suffered acute rheumatism two years before, and had dyspnœa ever since. On admission violent action of heart, mitral murmur, much pulsation of cervical vessels, pulse 136; child not greatly distressed. Given digitalis and paregoric. Did not keep bed till near her death; ate well; latterly had occasional fits of dyspnœa, pulse continuing excessively frequent, with no new symptom. Sank and died. Twenty-nine days in hospital.	Slight general œdema. The lower lobe and anterior depending tip of the upper lobe of the left lung in a state of red hepatisation. In the right lung precisely the same portions were in the same state, its lower lobe less uniformly affected than that part of the other side. Mitral valve covered with small, prominent, ragged vegetations, very soft, covering both edges of the valve. Partly decolourised clot in both ventricles. In the right kidney two small fibrinous blocks.	Old adhesions and a little fluid in both pleuræ; beneath the left a few spots of ecchymosis.
LXIV. 21. M. 28. 24.	Death so soon after admission that no history was obtained.	No œdema. Anterior edge of right lung a good deal solidified, as if from pneumonia, but not sinking. Front of left lung and almost the whole of its upper lobe consolidated, of brown colour, devoid of air, sinking, serous fluid in bronchial tubes. Heart, 1 lb. 5 oz.; one of aortic valves perforated by two ragged holes; left ventricle contracted. Kidneys enlarged and hardened. Liver and spleen enlarged and congested.	Fluid in pleuræ; in the left old adhesions.
LXIV. 115. M. 21.	Vigorous healthy man. Awakened one night by pain in chest and shivering, having been at cricket for a long time on the previous day (in March); perfect health	The right lung compressed by the distended pericardium against the back of the chest; the anterior portion of its upper lobe was of a dark colour, quite solid, and sinking in water; the	A few adhesions in each pleura. Pericardium

25.	previously. On admission dyspnœa, tubular breathing, &c., in left chest, and dull percussion; rusty sputum. Calomel and antimony every fourth hour. Gradual amendment. After six days, lung-sounds nearly natural; three days later erysipelas attacks chest, back, and shoulder; weak, irregular, faltering pulse. Bark and ammonia given. Lay on right side. Sank. Twenty-five days patient.	other parts of a pinkish colour, almost solid. Left lung compressed, but otherwise healthy. Heart healthy.	much thickened, and containing *two quarts* of pus.
LXIII. 265. M. 9. 26.	Delicate lad. Admitted with acute rheumatism. After eight days has all the physical signs of double pneumonia, mostly of right side. Makes a good recovery in about eight days more. Convalescence interrupted, seventeen days later, by return of pain in limbs and alarming attack of dyspnœa; pulse 120; visibly pulsating carotids; free respiration posteriorly. Soon up and about; well, or nearly so. Then, eleven days after the last, a sudden attack of angina, which carried him off in a few hours. Forty-three days.	All the right lung, except quite the apex, was red, solid, and airless, 'evidently in the first stage of pneumonia;' portions only sunk in water. Left lung was affected in exactly the same way, but less uniformly. The consolidation is scattered through the lung, so as to give to a section a marbled or mottled appearance. Mitral valve very much thickened by fibroid matter. Aortic valve slightly so.	Pericardium universally adherent by recent lymph. Adhesions of some standing in both pleuræ.

CLASS IV. *Table of Cases of simple Pneumonia.*

Reference to Post-mortem and Case-books.	Nature of case, &c.	Post-mortem appearances : site and extent of hepatisation.	Co-existence of pleurisy, or of pericarditis.
XLV. 200. M. 32. 1.	Well formed and in good condition. On day of admission seized with severe rigors, acute pain in right chest, and fever. When admitted skin is hot and dry ; face flushed ; pulse 108, not easily compressible. Bled to ℥xvj. Second day, pulse 120, small and weak ; very hot and thirsty ; a little brownish-red viscid mucous sputum. Cupped to ℥xvj., and given $\frac{1}{2}$ gr. of tartarised antimony every three hours. Third day, pulse 108, weak ; unable to obtain any rest, owing to vomiting caused by antimony ; is also much purged. Dose is reduced to $\frac{1}{4}$ gr. Fifth day, in very weak state, spitting mucus mixed with blood ; blistered. The blister rose well, but gave no relief. Herpes labialis now apparent. Jumped out of bed delirious. Soon after became insensible, and so gradually died. Six days in hospital.	The upper lobe of right lung and the whole of left lung 'remarkably healthy.' The lowest lobe of the right lung in various stages of red and grey hepatisation, adherent to the costal pleura by slender bands of lymph. All other organs healthy.	Recent pleurisy in connection with hepatisation.
LIV. 110. F. 18. 2.	Cough for a fortnight, with dyspnœa, pain in chest (earlier history not given). Admitted in state of considerable depression ; the face flushed, skin dusky, pulse weak, tongue dry and furred ; movements tremulous ; feeling of great weakness and lowness ; evidently unable to bear active treatment. She somewhat rallied subsequently (same day) ; but still there was	Body well made, somewhat obese. Besides the marks from bleeding and the vesication from the blister, some leech-bites were visible on the chest. There was a little fluid in the left pleura. The left lung was somewhat congested. The right lung was perfectly solid and non-crepitant throughout ; it gave out at its lowest part a turbid fluid as from grey hepatisation.	Right pleura obliterated by recent adhesions.

	thought necessary to give her wine. Second day, much hesitation felt as to propriety of bleeding. The pulse was weak and rapid, but the breathing very greatly oppressed. A small bleeding was made with great care. The pulse rather increased in power, and diminished in frequency the while. The blood was of course intensely buffed. Calomel and opium now given every three hours, and a large blister applied to the side. She died the following morning. Less than three days in hospital. [Verbatim report, with some curtailment, chiefly from omission of stethoscopic signs.]		
XLVII. 81. M. 32. 3.	A free drinker. Supposed to have been ailing some time with cough and symptoms of fever; but about his work till three days before; then rigors and pain, especially in right chest. On admission skin hot, tongue coated, rather dry; pulse 120, small and weak; aspect that of a man suffering from fever, headache and general pains. Eighteen leeches to chest; ¼ gr. tartarized antimony every four hours; calomel and opium thrice daily. Vomited the medicine. Third day after admission, tongue dry and coated; some wine given. (Blistered on second day.) Fourth day, delirium decided. Fifth day unconscious; passing motions, &c. involuntarily. Some brandy given. Continued to sink, and died on sixth day. Little expectoration, and that muco-purulent.	Upper lobe left lung grey hepatisation throughout, with specks as of commencing abscesses. Lower lobe compressed and dark red; portion only crepitant. Right lung loaded with red frothy serum; lower lobe in state of red hepatisation. Kidneys rather coarse.	Left pleura generally adherent by recent lymph at back part; much purulent serum, with flakes of yellow lymph. Right pleura contained small quantity of lymph, mixed serum and lymph adhering to lower lobes. Lymph and some fluid in pericardium.

Reference to Post-mortem and Case-books.	Nature of case, &c.	Post-mortem appearances : site and extent of hepatisation.	Co-existence of pleurisy, or of pericarditis.
XLVII. 95. M. 30. 4.	Three days before admission, rigors, headache, vertigo, vomiting, general pains, cough with white frothy expectoration ; no pain in chest ; no history of exposure. On admission, flushed dusky yellow, pulse 120, feeble ; tongue white and rather dry ; no chest-pain, even on deep inspiration. Second day, much heat of skin, and stitch in left side ; tongue dry ; pulse 120, sharp ; rusty pneumonic sputa. Third day bled to ℥xij. ; and same evening to ʒiij. ; tart. emetic. gr. $\frac{1}{4}$ every four hours ; blistered. Quite gradual typhoid sinking, and death on the ninth day.	Upper lobe of left lung perfectly solid and in state of gray hepatisation ; lower part equally solid with red hepatisation. Right lung generally crepitant, but at back gorged with frothy serum. Both kidneys rather large and slightly granular.	Left side, extensive recent lymph, the more recent at lower part. Right pleura not mentioned (healthy?). Slightly turbid serum (a little) in pericardium.
LV. 149. M. 25. 5.	A robust-looking man. Caught cold three days before admission ; aching in back, &c. ; cough and pain in the right side. On admission, face was flushed, skin hot, pulse hard ; there was pain on deep respiration at lower part of right side. Leeches, calomel, and opium. Two days later, the face was livid, the other signs remaining, pulse being more frequent. Leeches were repeated and medicines given every three instead of every four hours. Third day there was more pain at the upper part of the chest ; the expression was pinched and anxious. He was now blistered, and mercu-	Body in good condition, well made. The whole of the left lung was hepatised ; the front part was in a state of grey hepatisation. The lower part of the right lung was hepatised. The bronchial mucous membrane was vascular, with mucus in the tubes. Kidneys congested ; healthy. Other parts healthy.	Left pleura full of turbid fluid and shreddy particles. The membrane highly vascular. The diaphragmatic portion of the right pleura coated with recent fibrin.

	lower. Fourth day, wine was ordered, and he seemed to rally. Fifth and sixth days, he was very violent and noisy; then low typhoid delirium, and death. Eight days in hospital.		
LVIII. 64. M. 52. 6.	Good health till eight days before; then sore throat, cough, pain in left chest. Day or two after, pain shifted to right chest. It was severe at time of admission. Pulse 90, weak; respiration weak, hurried; (auscultation). Blistered over chest in front; cupped on right side. Antimony and nitre every three hours. Better till the third day, when he became very delirious. On fourth day pain in right side increased and friction was heard. Cupping repeated. Calomel, opium, and antimony every three hours. On the fifth day a large blister to the right side; difficulty in spitting. Death on the fifth day.	Left lung and pleura healthy. Lower lobe of right lung extensively inflamed, the whole of its tissue, except a small part of the outside, being infiltrated with a whitish-yellow deposit, which in parts had broken down into well-formed pus. Heart healthy, but for a little atheroma. Other viscera natural.	Pericardium distended with turbid fluid and coat of lymph, the whole membrane lined with it. Soft lymph in pleura, corresponding with hepatised lung.
LVII. 277. M. 35. 7.	Little or no history. Appeared to have been under treatment for pleuritic pain in left chest, for which he had been leeched and bled. Some account of a blow two weeks before. Suffering intensely. Less than one day under observation.	Lower part of upper lobe of left lung in a state of grey hepatisation; the apex healthy; the lower lobe much congested. The right lung crepitant throughout. Liver and kidneys healthy. Heart natural.	Left pleura quite obliterated. Surface of right lung coated with lymph half an inch in thickness. Some turbid serum.

Reference to Post-mortem and Case-books.	Nature of case, &c.	Post-mortem appearances : site and extent of hepatisation.	Co-existence of pleurisy, or of pericarditis.
LVI. 120. M. 30. 8.	A 'navvy,' who had been ill three days with catching pain in the right side and short hacking cough. Previously quite well. Face flushed ; pulse 96, feeble ; tongue coated ; respiration very short and hurried ; crepitation of various degrees all over back of right side, and universal dulness. Salines and antimony. Bleeding on second day. On fourth day, renewal of dyspnœa and much pain in left side. Antimony omitted. Great exhaustion from this time ; and he died on the fifth day.	Body somewhat emaciated. The right lung completely hepatised and solid ; nowhere crepitant. The bronchial tubes occupied by light-coloured coagula. The left lung was in a similar condition ; but the lower part of it was only to a slight extent affected, much of it being spongy and natural. Heart natural. Capsules of kidneys somewhat adherent ; the organs otherwise natural.	Tolerably recent adhesions to some extent, on both sides of chest. Some turbid fluid in left pleura. Numbers of shreds of fibrin in the pericardium.
LX. 74. M. (?) 9.	Formerly a coachman. Good health till he got wet six days before admission. This was followed by rigors and dyspnœa. Admitted blue, gasping, and speechless for want of breath ; the extremities cold ; dyspnœa did not diminish. Died in the evening, quite sensible to the last. Death almost entirely by apnœa. Seven or eight hours in.	Back of the right lung in a state of grey hepatisation as to its upper and middle lobes. The opposite lung congested, but crepitant. Structure of kidneys confused, but nothing more. Other organs healthy.	Uniform membranous layer of lymph, on both sides, over nearly all the pleural membrane.
LXII. 148. M. 62. 10.	A labourer. Attacked suddenly on his way to church, five days before admission. Giddiness and shivering ; pain in left side ; dyspnœa. In very weak state on admission. Pulse 100, very small ; hands and	Good condition. Lower lobe of left lung in a state of grey hepatisation ; pus could be squeezed out of it. The bronchial tubes were very red. The right lung healthy.	Left lung surrounded by large quantity of lymph. Old adhesions on

	little ; then became delirious, dyspnœa more urgent. At noon of third day was dying, respiration extremely rapid. Died gradually. Three days and a half in.	other viscera healthy.	quantity of fluid in pericardium.
LXIV. 128. F. 50. 11.	Admitted moribund. Said to have been ill about five months. Died in a few hours.	All right lung, except a very small piece at its apex, converted into a mass of grey hepatisation, very granular in fracture, and pouring out pus abundantly on pressure ; its weight, $3\frac{1}{2}$ lbs. Weight of left lung, natural, $15\frac{1}{2}$ oz. Liver slightly fatty. A small cyst in right ovary. Heart, &c. healthy.	On right pleura, between lung and diaphragm, much gelatinous lymph.
LXIV. 117. M. 28. 12.	A single man of dissolute habits ; in failing health for three years, and often laid up with lung-symptoms. In his usual health morning before admission ; went to work at 5 A.M. : at 10 A.M. brought home in violent fit of rigor ; put to bed ; soon began to ramble, and then to be violent, requiring to be kept in bed by force. On admission, continued to ramble ; with difficulty kept quiet ; bowels very loose ; tongue dry and brown ; breathing rapid ; no orthopnœa ; neither cough nor sputum ; dulness, tubular breathing on right side. Opiate enema ; calomel and antimony ; brandy. Soon obstinately refused medicines. The bowels continued rather loose ; much raving. Fourth day, he began to sink ; occasional returns of raving ; respiration very hurried. Five days in hospital. (Six days ill.)	The left lung had some patches of ecchymosis on its surface ; part of its upper lobe was congested, yet contained air, and floated. Weight of left lung 18 oz. The right lung as solid and unyielding as liver. It occupied all the pleural cavity, and was entirely hepatised, save a very small patch close to its lower edge, which still contained air ; elsewhere the lung was quite airless. The entire organ sank. The upper lobe was of an uniform buff colour, infiltrated with pus. The lower lobe was browner. Everywhere the texture was remarkably granular. Weight of right lung 3 lb. 11 oz. Kidneys large, smooth, coarse. Transparent coagula in heart's cavities. Liver large ; its surface very uneven ; capsule thickened ; the tissue gathered into sphericles, with large quantity of fibrin matter between them.	On left pleura, a small quantity of serous fluid. Both layers of right pleura coated with recent lymph.

Reference Post-mortem and ase-books.	Nature of case, &c.	Post-mortem appearances : site and extent of hepatisation.	Co-existence of pleurisy, or of pericarditis.
XIV. 36. M. 23. 13.	Ill for a month ; pain and lumps about larynx, and dysphagia ; and then pain under left nipple (not 'stitch'). History very imperfect, from his want of voice, and the pain which speaking caused. Stout ; manner natural (auscultation, signs of left pneumonia) ; sputum rusty ; partial orthopnœa ; face venous. Second day, pulse 80. Third day, pulse 88 ; distressed breathing ; has had severe pain in left side and epistaxis. Six leeches, calomel and opium. Died on fourth day, after two severe epileptic fits, ten minutes before and at death, respectively. Four days in-patient.	Upper lobe of left lung much congested ; its lower lobe in a state of grey hepatisation, exuding pus-like fluid. Lower lobe of right lung congested. Cortex of kidney increased (natural appearance under microscope). Other organs healthy.	Left pleura contained a pint of purulent fluid. Recent lymph on surface of lung. A little clear fluid in the right pleura.
XIV. 161. F. 47. 4.	Ill two weeks with cough and pain in left side. These came suddenly with rigor. The sputum at that time 'like currant-jelly,' and some unmixed blood. On admission, much distressed for breath ; frequent pulse ; much thirst ; cool skin. Left side dull. and vocal fremitus absent ; blowing breathing. Respiration became worse, and she died. Rather over one day in the hospital.	Lower lobe of left lung completely hepatised, so as to sink as a whole ; it had a greyish-brown colour, and a finely granular fracture. Posterior part of right lung in a state of early hepatisation. The small bronchial tubes were packed full of very thick muco-purulent matter. The muscular fibres very conspicuous. Heart natural ; left ventricle imperfectly contracted.	Thin layer of recent lymph in the left pleura.
LXIV.	Admitted moribund. No history.	Right lung much congested ; especially its upper lobe, which was nearly solid, and contained	Sub-pleural extravasation

		Yellow coagula in right ventricle.	
LXIV. 231. M. 23. 16.	Seized with shivering and febrile symptoms two weeks before; kept bed four days; has much the aspect of fever; pulse 132; skin warm; a little thick, bile-stained sputum; averse to food; delirious. Calomel and opium every four hours. Second day, 6 oz. of port-wine; blister; delirious and violent during night; hot skin and tympanitic belly; respiration quickened, but no distress from dyspnœa. Pill stopped third day. Continued to sink till death. Four days and a half in-patient.	Emaciated. Tinge of yellow on skin and conjunctiva. Whole of right lung in state of grey hepatisation; as solid as liver, and exuding much pus; sinking as a whole. Weight 2 lb. 10 oz. Left lung natural; weight 12 oz. Liver congested in patches. Liver cells opaque, granular, of a faint brown. Other viscera natural.	Small quantity of purulent fluid in left pleura. On right, some clear fluid and some recent adhesions. Recent lymph in pericardium.
LVII. 304. M. 28. 17.	Represented as quite well three days before admission. Drank neat gin largely on the 26th December. Breathing was affected on the 27th. Was admitted on the 28th, when his manner was quiet and natural; pleuritic friction was then audible, and mucous râles masked the sounds of the heart. Antimony, calomel, and opium. Night of admission had restlessness, which soon passed into furious delirium. Two days in patient.	Lower lobe of right lung in a state of red hepatisation. The upper lobe in a state of grey hepatisation. Some emphysema at edges of both. There was also bronchitis. The vessels of the lungs were plugged with lymph. Liver congested. Kidneys healthy. Brain not examined.	Recent lymph in both pleuræ and in pericardium.

CLASS V.

Table of Cases of Hepatisation where death was ascribed wholly or mainly to low or latent Pneumonia.

Reference to Post-mortem and Case-books.	Nature of case, &c.	Post-mortem appearances: site and extent of hepatisation.	Co-existence of pleurisy, or of pericarditis.
XLVIII. 262. M. 33. 1.	On admission presented all the appearance of an ordinary case of fever, extreme deafness having come on at time of illness two days before; the other symptoms being shivering, headache, sickness, dyspnœa. On admission he was very dirty, p. 120; had headache and much wandering (attention not called to the chest, which was not examined till six days later); had some cough, with scanty adhesive sputum; urine was retained in bladder; delirium occurred; face became livid, and cold sweats occurred. Death, by gradual sinking, on the twelfth day after admission.	Dark serous fluid in left pleura. Posterior part of left lung was much congested and solidified; the whole of right lung was quite hepatised, firm and solid; small deposits of pus in this lung. The other organs healthy.	Some recent lymph at base of right lung.
LII. 173. M. 39. 2.	Reported of very dissipated habits, and to have had attack of 'inflammation of stomach' (there was mark of blister on right side of chest), for which calomel and opium had been given. On admission was very restless and excited, so that he could hardly be kept in bed; state like delirium tremens. After two or three doses of laudanum, he slept for two hours; woke more collected; his face, however, was now dusky, and breathing oppressed. He sank very rapidly, having been about a day	The upper lobe of right lung solidified completely, light grey fluid escaping on section; supposed to be in early stage of grey hepatisation; the lower parts of lung more solid than natural. On left side, lower part of upper lobe the same as lower lobe of right; the bronchial tubes very vascular. Kidneys congested. Other organs natural.	Pleural cavities natural.

LIII. 62. M. 27. 3.	A history of having caught cold, and having been ill in consequence for three or four months. The illness ascribed to standing in water. Four days before admission, acuter symptoms. Was admitted much emaciated, with rattling sounds in bronchial tubes. About two days in hospital.	The right lung solid; reddish grey *as to its upper lobe*; other parts of lung very congested. On left side, the lower lobe in the same so-called hepatised state; the upper part congested. Much thick mucus in the bronchial tubes.	Some firm pleural adhesions on both sides, but no lymph.
L. 114. F. 64. 4.	Nine days ill, the main feature being great debility and prostration, with but little cough. Sunk after seven days.	Upper lobe right lung consolidated and grey, approaching to suppuration. The left lung and lower of right lung emphysematous.	A few old adhesions only.
LIII. 66. M. 17. 5.	Of remarkably bloodless expression. Ailing about sixteen months, 'some slight illness' from which he had not recovered. No cause could be made out for his condition. Some hæmoptysis occurred (amount not stated) seven days after admission; great prostration followed. He died suddenly the following day. [No record of examination of urine during life. It was believed that it had been tested, and found healthy. After death, that found in bladder was distinctly albuminous.] Eight days in.	Exceedingly exsanguine hue noticed at autopsy; the blood very fluid; all viscera exceedingly pale, but otherwise natural. Lungs very œdematous at apices. In middle and lower portions exceedingly consolidated, and of a dark reddish-brown colour, sinking in water. The heart natural.	Pleuræ and pericardium natural.
LIV. 115. M. 45. 6.	A Frenchman, from whom an imperfect history was obtained of eleven days' illness, viz. pains in limbs and bilious vomiting, the pulse being 'pretty quiet;' no chest-pain. From yellow he gradually became jaundiced, got cough and muco-purulent expectoration. Died after eleven days. (No diagnosis of pneumonia.)	Whole of right lung, except the base, inflamed; the texture infiltrated with sero-purulent fluid. The left lung emphysematous in front, congested and œdematous behind. Kidneys large and congested; their capsules rather adherent.	Recent cavity in right pleura, with thick recent lymph.

Reference to Post-mortem and Case-books.	Nature of case, &c.	Post-mortem appearances: site and extent of hepatisation.	Co-existence of pleurisy, or of pericarditis.
LV. 209. M. 62. 7.	Much emaciated; a year out of health. Seven weeks lumbar pain, œdema of legs, vertigo. and great debility. On admission, urine pale and albuminous; vomiting frequent Fourth day, diarrhœa; afterwards gradually sank. Ten days in hospital.	Body in good condition. Œdema of legs. Lower part of right lung in a state of grey hepatisation. Lower part of left lung slightly hepatised. (Kidneys too much decomposed for examination.)	Sero-purulent fluid in right pleural sac. Pericardium and heart natural.
LVII. 20. M. 42. 8.	A butler of good general health till five weeks before admission, when he had a 'cold' and dyspnœa. At this time he was knocked down by a severe blow on the left thigh; considerable swelling and partial loss of power of that limb followed. There were no symptoms to indicate chest-mischief. Fifth day he had severe rigor, followed by great collapse. Seventh day, a second rigor and collapse. Purulent urine. Death on the eighth day.	Left leg greatly enlarged. The whole of the left lung hepatised; one or two masses of fibrin in its upper part. The same condition, to slighter extent, in upper part of right lung. Femoral and popliteal vessels, and their branches, occupied by dark-red fibrin. The cavities of the heart dilated.	Softish rough fibrin over the pericardium; old cystitis. Left pleural sac obliterated.
LVII. 43. F. 54. 9.	Admitted for old sloughing ulcers; began to complain of sternal pain; stomach was leeched, &c Lingered on, and became almost comatose a day or two before death. Thirty-four days in-patient.	Grey hepatisation of upper and lower lobes of right side. Left lung emphysematous. Kidneys small, and with adherent capsules.	Recent pleurisy of right side. Left pleura healthy. A little turbid fluid in the pericardium.
LVII. 176.	On admission, very much exhausted and very	Body well formed.	Firm pleural

F. 24. 10.	dirty; diarrhœa and vomiting for past three weeks. At present headache, brown tongue, weak pulse, a loud systolic murmur. Given astringents and prussic acid. The urine slightly albuminous; sensations referred to the abdomen. She lay groaning and calling out night and day. Fifteen days in hospital.	Posterior flap of mitral very much thickened. Heart otherwise healthy. The upper lobe of right lung much inflamed; its posterior part in a state of red hepatisation. The lower lobes and the opposite lung much congested, infiltrated with fatty serum. Spleen large, 16½ oz. Left kidney rough, small, mottled; diminished cortex. Other organs healthy.	adhesion right side.
LVIII. 37. F. 29. 11.	Admitted with extreme debility, after child-bearing one month before. Rigors had occurred daily since that event; a dry cough and profuse sweating. Given bark, brandy and stimulants. Thirteen days in.	The body not emaciated. Lower lobe of right lung in a state of grey hepatisation; its tissue in part broken down, so as to form small abscesses. The opposite lung contained several masses of extravasated blood, also breaking down in parts. Kidneys pale and smooth, with stellate veins. Uterus as usual a few weeks after parturition. Heart large, with healthy valves and firm white clots in its cavities. Spleen almost diffluent.	Fluid and recent lymph in right pleura. Left pleura healthy.
LIX. 131. M. 42. 12.	Had sore-throat three months before admission. No history of syphilis obtained. The man slow and muddled and hesitating in speech; left pupil rather the smaller throughout; distinct loss of power in both hands; pulse 112. Given ammonia, brandy; later, iron and calumba. Gradually lost memory completely, and got delusions of all sorts; violent at night. Two days before death became comatose; pupils very contracted; pulse gradually accelerating up to 160. Ten days in hospital.	Emaciated. Slight softening of corpus callosum and septum ventriculorum (whether post mortem, or otherwise, doubtful). Lower lobe of right lung in a state of red hepatisation; rest of lung spumous, as was the left. Heart and remaining organs healthy.	Pleural adhesions at left base and right apex.

Reference to Post-mortem and Case-books.	Nature of case, &c.	Post-mortem appearances : site and extent of hepatisation.	Co-existence of pleurisy, or of pericarditis.
LIX. 164. M. 39. 13.	A great drunkard ; suffered delirium tremens more than once. Out of health now for two weeks, with lassitude, headache, and pain in the limbs. Gave up four days before admission, and took to bed. Admitted with muttering delirium ; not violent, but fidgety and restless ; skin cold ; pulseless ; no spots. Nape of neck blistered ; wine and ammonia given. Died in a few hours.	Body in good condition. Right side of heart filled with decolourised clot ; extensive old pleural adhesions on both sides. Left lung healthy. The whole of the two upper lobes of the right in a state of grey hepatisation, sinking in water. Bronchial tubes full of mucus. A few cysts on the surface of the kidneys ; their capsules adherent.	Old adhesions both sides. Pericardium natural.
LIX. 193. M. 48. 14.	Admitted dying. A hard drinker, who had had repeated attacks of delirium tremens.	Heart dilated and flabby, apparently fattily degenerated. Lungs much congested ; lower lobes on both sides inflamed and very friable. Liver and kidneys healthy.	Pleuræ and pericardium natural.
LXII. 208. F. 36. 15.	Admitted July 28. A week before, seized with shivering and acute catching pain in right side. Cough came on about the same time, and, with dyspnœa, had continued ever since. On admission, aspect distressed and anxious ; face flushed ; respiration 52 ; pulse 120, and very weak ; skin hot and dry ; tongue dry and brown ; lay towards the left side. In right side of the chest breathing was very harsh and imperfect ; no ægophony or tubular breathing. A few moist sounds were heard at the apex on the left side ; breathing was free. Both sides were equally resonant. Ordered antimony wine in ammonia saline and port-wine	The body was plump and in good condition. There were no spots on the skin. The brain was perfectly healthy in all respects. The whole of the right lung was in a state of grey hepatisation, except a small portion near the apex and a small patch at the lower sharp margin. The texture was grey and mottled, and in parts softened as if on the point of breaking down. Pus could be readily squeezed out. The bronchial tubes were congested, and contained frothy fluid. The left lung was healthy, except that it contained near the apex two minute cretaceous tubercles. The left ventricle was uncontracted ; it contained fluid blood, with two or three little pieces of	The right pleura was natural. About the left there were a few old adhesions.

	(℥vj.) daily. Last day, much the same; face still anxious; a slight cough, but no spitting. Ordered ammonia salines; port-wine ℥xvj., daily.—30th. Quite delirious; respiration not so rapid; pulse 120, with some power; tongue dry, brown, and chapped. In front of the chest on both sides air was admitted, but imperfectly on the right, and very harsh was the breathing that was heard. A few small indistinct spots (supposed to be the mulberry eruption of typhus) appeared on the belly.—31st. Delirium continued; gums and tongue covered with sordes; respiration between 50 and 60; pulse 120; skin very hot; face flushed; the eruption just as it appeared the day before. Ordered 3 ℈j. doses of quinia in three successive hours; afterwards two grains of quinia every four hours.—August 1st (twenty-four hours after the quinia was ordered). The skin was quite cool; pulse 72; respiration 60 a minute. She put out her tongue when told, and helped herself to some water. The next instant, within half a minute of the pulse being taken, the colour faded from the cheeks, and she was dead. About four days in-patient.	black coagulum. The right ventricle was also uncontracted; it contained a decolourised clot of great length and tenacity, extending into the auricle and cavæ. There was a trifling amount of atheroma on the aorta and the mitral valve. The spleen and supra-renal capsules were soft. Kidneys and liver healthy. Large and small intestines perfectly natural. Uterus contained a little purulent matter.	
LXIII. 125. M. 33. 16.	He and his wife said to have been lying ill a fortnight. His appearance that of a fever-patient, but no eruption; flushed; semi-comatose; dry parched tongue; hot dry skin. Died without change. Ten hours in.	Signs in brain as of a very old clot. Upper lobe of left lung in state of red hepatisation, approaching grey. At the back of the left lung a patch of pulmonary apoplexy. Thin layer of recent lymph in the heart, and beneath it old lymph deposited. Peyer's patches congested. The kidneys granular.	Adhesions (old ?) in right pleura.

Reference to Post-mortem and Case-books.	Nature of case, &c.	Post-mortem appearances: site and extent of hepatisation.	Co-existence of pleurisy, or of pericarditis.
LX. 5. M. 42. 17.	Admitted in a state resembling fever. Said to have been losing flesh, and out of health for three months; had shivering ten days ago, and, shortly after, a fit like epilepsy. On admission, pulse 80; general state like that of phthisical patient with fever; no cough or expectoration, or pain at chest. Lay picking at bed-clothes. Two days' residence.	There were a few small unsoftened masses of crude tubercle at the apex of the left lung; remainder of that lung was quite healthy. The whole of the right lung was hepatised, except a layer an inch thick at its lowest part. At the upper lobe the hepatisation was grey; lower down, red. Decolourised clot occupied the right side of heart. Kidneys finely granular; capsules adherent; cortex somewhat diminished.	Recent lymph was smeared over the right lung.
LX. 108. M. 52. 18.	A free liver, who had long suffered from eructations and epigastric pain. The day before admission, he was thrice attacked with hæmatemesis, each time vomiting about half a pint of blood. On admission, had slight cough with mucous spitting. Bronchial sounds were heard about the chest, and the liver was felt enlarged somewhat. Gallic acid and stomachic medicines were given. He got better after eleven days, and took an airing. The evening after, had a rigor, with great depression and want of breath. He lay from that time on his right side, evidently dying. No treatment was attempted, save stimulants. Fifteen days in hospital.	Rather emaciated. A patch of consolidation middle of back part of left lung, and another near the apex. In these parts the lung was very spumous, contained a good deal of blood, and only just floated. Whole of right lung in a state of grey hepatisation, except a very small part at the base. Other organs healthy, save kidney, which had two cysts on its surface, in one of which was coagulated serum having a whitish succulent mass of fibrin.	Extensive old pleural adhesions on both sides.
LXI. 92.	Suffered privations for three months, and for as long had pains in the loins extending	Heart healthy, but for slight atheroma; right ventricle and pulmonary artery full of deco-	Right pleura obliterated.

M. 65. 19.	down the left leg. He was much emaciated. The pain continued, and was very acute. Two days before death, severe cough came on with viscid dark-brown sputa. The day before death there was much pain in the left chest, and dyspnœa. Calomel gr. iij., and opium gr. j., every three hours. Thirteen days patient.	lourised clot; red blood-clot in the left ventricle. Right lung healthy. Nearly all lower lobe of left in a state of grey hepatisation. Double inguinal hernia. Remaining organs healthy.	A very large collection of purulent serum in the left pleura, and recent lymph smeared on the surface of left lung.
LXIV. 186. M. 35. 20.	Epilepsy; delirium; aspect, &c., like that of fever. Died slowly, exhausted. Five days in hospital.	Lower lobe of right lung consolidated as in first stage of pneumonia. Congestion of brain-substance. Other organs appeared quite natural.	(Caret) healthy?

CLASS VI.

Eight Cases where, from defect of history, the details do not suffice for classification.

Reference to Post-mortem and Case-books.	Nature of case, &c.	Post-mortem appearances: site and extent of hepatisation.	Co-existence of pleurisy, or of pericarditis.
LVII. 39. M. 32. 1.	A clerk, who had been out of health for some months; latterly he had 'caught cold,' and for a week kept his bed; later (exact periods are not given in the Report), acute pain occurred in the left side, like 'stitch.' Was admitted two days after, with pinched features, in great distress from dyspnœa. On auscultation fine crepitus, &c. Given calomel and tart. antimony every two hours; blistered; some wine occasionally. Wandering; death. Under four days.	Body in good condition. The lower and posterior parts of both lungs in a state of hepatisation. Lining of bronchial tubes very vascular. Kidneys large, but smooth. Heart natural.	Old adhesions of both pleuræ. Recent soft fibrin in both. Pericardium natural.
LVII. 93. M. 35. 2.	A potman, who had had cough and hæmoptysis for three or four months. Admitted unconscious, very prostrate, hot skin; condition like continued fever, but no spots; urine collected in bladder. Thirty-six hours in-patient.	Middle lobe of right lung in a state of grey hepatisation. The lower lobe was red, softened, and breaking down. One-third only of the whole lung floated. The liver was large, mottled, and greasy. Kidneys large, coarse, healthy. Heart was large.	Old adhesions in left pleura. Right pleura universally adherent. In front and at upper part these adhesions were recent; posteriorly lower down they

			were jelly-like, apparently older.
LVII. 95. F. 3. 3.	Admitted in a state like scarlatinal dropsy. Unhealthy aspect, large head, &c. Three weeks ill, described as 'a cold.' Large quantity of bronchial mucus. No examination of urine.	Bronchial tubes inflamed, and full of frothy mucus. The lung in various parts solidified, *e.g.* at both apices. Fatty liver.	Pleural cavities natural.
LXIII. 20. F. 40. 4.	(Imperfect history from her husband.) Sticking to her work (though falling down from 'pain' and exhaustion) till four days before admission. On admission, dulness of right side, &c.; the patient dusky, and quite exhausted. Soon died. Under two days. (Lay low in bed; respirations very rapid and shallow; scarcely, if at all, conscious.)	Belly tympanitic. The whole of the lower lobe of right lung in a state of red hepatisation, verging on grey; the whole sinking. Left pleura and lung healthy. Liver slightly fatty. Kidneys slightly rough on surface. Other organs natural.	Fluid in right pleura.
LVIII. 145. M. 30. 5.	Cough for eight days before admission, followed (in two days) by rigors and pain in the left side; anxious; face flushed, skin hot, pulse frequent. Auscultation. No albumen in urine; rusty sputum. Antimony and calomel. Twelfth day revived somewhat, and was given wine, bark, &c. Rather sudden relapse and unexpected death. Twenty-two days in hospital.	Whole of back of left lung hepatised; the right lung somewhat congested and œdematous. Kidneys rather large, with adherent capsules.	Purulent fluid and honeycombed lymph in the pericardium. Substance of heart dilated and soft. Lining membrane blood-stained. Pleuræ not mentioned.

Reference to Post-mortem and Case-books.	Nature of case, &c.	Post-mortem appearances : site and extent of hepatisation.	Co-existence of pleurisy, or of pericarditis.
LXII. 104. F. 28. 6.	History of rigors and pains in the limbs thirteen days before admission ; then cough and bloody sputum, the quantity of which 'had decreased very much.' Was told she had fever. Admitted with acute symptoms, hot skin, flushed face, husky voice. Ordered wine. Expectorated a very little dark-coloured sputum. Second day, on auscultation, friction was heard (pleural or pericardial ?). Pulse 120. Ordered calomel and opium, and wine increased. Third day, pulse more frequent. Left side was blistered. She soon tore the blister off, and talked wildly. Dyspnœa was now urgent. Wine and brandy given. She gradually sank, and died the next morning. About four days' residence.	Lower lobe of left lung perfectly solid, and devoid of air. Of a light fawn-colour and compact texture. The upper lobe quite natural ; the transition from one to the other quite abrupt. Slight emphysema of the right lung. One kidney dwindled into mere fibrous tissue. The other healthy, pale.	A little straw-coloured fluid in the pericardium ; and a little recent lymph on the surface of the heart. Pleuræ not mentioned.
LIII. 254. F. 22. 7.	Ill only a week, with cough and sore-throat, with pain in left side. On admission, face very dusky ; breath very hurried ; pulse 130, full, firm. Bled to 10 oz. Auscultation next day ; coarse, moist sounds universally, but no dulness. Blood not buffed nor cupped. The expectoration scanty and purulent. On the 5th day there was some approach to fine crepitation or friction at lower part of left lung without marked dulness. She sank gradually, with typhoid symptoms. Nine	Right lung healthy and crepitant throughout, but the bronchial tubes of both lungs vascular, and full of muco-purulent fluid. Back part of lower lobe of left lung solid and compact, breaking down under pressure. Gall-stone blocking up cystic duct.	A single band of adhesion in left pleura.

F. 11. 8.	dyspnœa, and death in a few hours. Twelve days. (Had had acute rheumatism some years before, and cough for two months. Frequent pulse, hot skin, and throbbing carotids noticed before the sudden collapse of which he died.)	right lung, were patches of lobular pneumonia as big as peas. Old thickening and recent blood-stained lymph on the cardiac valves.	pericardium. .

APPENDIX E.

ABSTRACT OF THE CASES OF 43 YOUNG CHILDREN EXAMINED AFTER DEATH, AND EXHIBITING LUNG-CONSOLIDATION REGARDED AS PNEUMONIC.

TABLES similar to those of the foregoing Appendix I have constructed in reference to children. Of these space will allow but an abstract.

From July 1870 to the same month 1875 there occur out of a total number of 278 children examined after death at the Hospital for Sick Children, Great Ormond Street,[1] 143 exhibiting lung-consolidation variously disposed, and generally described as being pneumonic. These may be divided in the first instance into 40 cases where the lung condition followed upon anterior disease, and was not of itself or alone the cause of death, and 3 where it was acute and recent.

Classifying first the 40 we find

5 associated with cardiac valvular obstruction.
5 ,, ,, scarlatinal dropsy.
5 ,, ,, rickets.
4 ,, ,, typhoid fever.
3 ,, ,, diphtheria and croup.
3 ,, ,, measles.
3 succeeding pericarditis.
2 had in addition miliary tubercle.
2 were associated with bronchitis of long standing.
1 was a gangrenous excavation, mistaken in life for empyæma.
7 were secondary to long-standing disease, presenting no active symptom, the state of the lung in most instances overlooked.

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These several classes may be rehearsed as follows :—

I. In the 5 cases of *cardiac obstruction*, the pulmonary consolidation was double in all, in one of these (a child of 2½ years) it was described as 'lobular.' The lungs in two instances were 'tough and leathery.' There was pleurisy in two and recent pericarditis in one. In every instance the heart affection had been first observed, and the state of the lungs had apparently arisen out of it.

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[1] The total number of children admitted during these five years exceeds 4,000.

II. In the 5 cases of *scarlatinal dropsy*, hepatisation was double in 3, and in 2 of these 3 'lobular.' There was pleurisy in none, old pericarditis in one. In the two cases in which the lung affection was single it had not advanced to solidity, but the lung state is described as 'commencing pneumonia' in one, and as 'inflammatory engorgement' in the other. In all the cases general dropsy was the symptom which claimed and obtained treatment.

III. In the 5 cases with *rickets*, consolidation was double in all, and lobular in 3 or perhaps 4. There was neither pleurisy nor pericarditis in any. In three of the cases portions of the lung are spoken of as 'carnified, shrunken, collapsed.' The 5 cases hang well together. The children, besides being rickety, are emaciated and poverty-stricken ; they die slowly of bronchitis, with considerable dyspnœa ; there is little or no elevation of temperature, thus in one instance (56, vol. ii.) the respirations are 74, while the temperature is 99·8°.

IV. In the 4 cases with *typhoid fever*, consolidation was double in 3, and lobular in one of these 3. *There was no pleurisy in connection with the consolidation in any*, but in one instance ('imperfectly examined owing to the presence of the child's father') the right lung is 'congested and bound down by recent adhesions,' while it is the *left* lung which is hepatised. Death in this instance occurred a month after the first symptoms of fever, and 4 days after active lung symptoms, the ulceration in the ileum 'apparently healing.' Conceivably it is a case of double pneumonia *after* typhoid.

V. In the three cases with *diphtheria and croup* the diseased condition (which was double in one) was hardly to be called consolidation. In one (where there was scarcely any dyspnœa throughout) the right lung was partially collapsed, non-crepitant, and sinking, owing to diphtheritic occlusion of the bronchus leading to it. In another there were similar patches of collapse in the right lung, together wiih 'lobular pneumonia in the stage of red hepatisation,' successive steps in the same process (see Chap. IX. p. 128). In no case was there pleurisy or pericarditis.

VI. In the three cases of *measles* the consolidation is both double, and lobular in one (an infant of 11 months, where bronchitis succeeded to measles, and purpuric spots appeared on the face). In one, a boy of 5, who had in succession typhoid, scarlatina, and measles, the consolidation was single, and accompanied with recent pleurisy; it presented indeed the appearance of pneumonia. In the account of the case, which is too brief. it is said merely, in reference to its close, that the child 'died of measles.'

VII. In the three instances where hepatisation *followed pericarditis*, two were single and one double. The two had recent pleurisy, in the other the lungs are bound down by old adhesions. In pyrexia and duration these cases, notwithstanding the antecedence of pericarditis (which seemed to determine the fatal issue), resemble pneumonia. The youngest of these three children was only 2 years and 4 months old. Here the pericardium was distended with eight ounces of purulent fluid containing lymph, the upper lobe of the *right* lung was consolidated, the middle lobe in part collapsed, and the lowest 'tough and carnified.' The pleura contained recent lymph. The bronchi were intensely congested.

Of the two instances of *tuberculosis*, one records, in a child of 6, firm greyish-red pigmented consolidation of the upper and middle lobes of the right lung, while the lowest contains a number of grey granulations, the bronchial glands being enlarged, and one of them caseous. The history is of chronic illness; six months' wasting, with cough and blood-tinged spitting. The other, a rickety child of 3½, died exhausted by diarrhœa. The lungs were very œdematous, and in spots collapsed. A few tiny granulations were scattered about them both, but no tubercle could be found elsewhere. The brain, however, was not examined.

There are two cases of *chronic bronchitis*, exhibiting diffused consolidation (alveolar catarrh) in both lungs, associated with collapse, without pleurisy and without-tubercle.

There is a case of *gangrenous excavation*, mistaken in life for empyæma, where, as usual, consolidated lung surrounds the cavity.

Lastly, as with the adults, we have seven cases of lung-consolidation in connection with prolonged disease, where the lung changes give rise to no active symptoms, but the patient gradually wastes, with little or no pyrexia. These include instances of cerebellar tumour, of hemiplegia, of mere wasting unto death from an undiscovered cause in a girl of three, of hereditary syphilis, and so on. In most of the cases the emaciation is extreme, and the low vital condition is indicated by bedsores.

Thus the instances of fresh pneumonia are reduced to three, or rather to two, for one of the three ought properly to be included with those mentioned above, of pneumonia following upon pericarditis. It has been mentioned already, that in this association the symptoms and course still resemble true pneumonia. If, therefore, we include the whole of these, the total number of cases would be brought up to six. At the same time, in estimating the *mortality* of pneumonia in children, it is obviously unfair to include instances of pericarditis.

Pneumonia pure and simple, as these statistics inform us, is very rarely fatal to young children.

Taking the six cases together, subject to this observation, the best marked is that of a well-nourished, healthy child, nine years old, who died after six days' illness, with dyspnœa for the last three days only. The earliest symptom was nausea with vomiting, and soon a hacking cough. On admission, two days before death, dyspnœa was urgent, and with orthopnœa and much restlessness, the face being cyanotic. No pericardial rubbing was to be heard, and the physical signs indicated consolidation of the left lung. The condition of restlessness and dyspnœa continued up to the child's death. On post-mortem examination both lungs were found covered with opaque corpuscular lymph, $\frac{1}{4}$ inch or more in thickness. The external surface of the pericardium was also covered with lymph, and within it was serum, with lymph shaggy and villous. The left lung as to its lower lobe was solid and red, with signs of grey mottling in one or two places. The upper lobe of this lung and the whole of the right lung were greatly engorged, and the lower half of the inferior lobe of the right was collapsed, and sank in water. The left lung weighed 11½ oz., the right 9¼ oz.

In a second case, that of a girl of ten, chorea of three weeks' standing had been followed by pericardial rubbing and signs of pleurisy of the left side. The child died suddenly, with blue face and jactitation, as one asphyxiated. On inspection the pericardium was found adherent by recent lymph, as was also the left lung. Some fibrin coated the aortic and mitral valves. There was no absolute solidity on the part of the lungs, but both were congested and œdematous, and there was pleurisy in connection with the left. An exactly similar sequence, viz., chorea, pericarditis, marked consolidation in both lungs, which were bound down by old adhesions, occurred in a girl of eight. She also, with paroxysms of dyspnœa, died like the others partly by asphyxia.

The third case is described as double pneumonia, but is somewhat imperfect. The lower lobe of the right lung was 'solid, firm, and somewhat tough;' there was some recent lymph on the surface of the *left*. The child (aged two and a-half) was ill 12 days in all; it was 'at times short of breath;' pulse 136, respirations 48. Some time before death it became unconscious. The child, it may be mentioned, was bled to one ounce without relief.

Strictly speaking, then, there is but one case of simple pneumonia, the others being anteceded by pericarditis, yet this association does not appear greatly to modify the phenomena of the disease, though it may suffice to render it fatal.

From the whole number of cases (of which a few details only have here been given), the following conclusions, amongst others, may be deduced.

First, that true pneumonia, as a fatal disease, is very rare in childhood.

Secondly, that the association with pericarditis, already noticed in the adult (p. 80), is more common in early life.

Thirdly, that the secondary lung-consolidation of childhood is very intimately connected with pulmonary collapse, the connection being most frequent and obvious in infancy.

The first conclusion seems opposed to that of a high authority upon children's diseases, my esteemed colleague Dr. West, who asserts that lobar pneumonia is often met with in early life. Yet when to that statement is appended others by the same writer, as that in infancy double pneumonia 'preponderates greatly' over those where one lung only is involved, that there is 'a tendency to pass into gangrene,' and that in 13 only out of 115 cases was inflammatory lung disease an idiopathic affection, it will appear, not that my conclusions of fact are different from his, but that Dr. West regards that as pneumonia which, rightly or wrongly, I have here excluded from that category. (See West, on 'The Diseases of Children,' Chapter xx.)

APPENDIX F.

THE ASSOCIATIONS OF PULMONARY GANGRENE.

THE statement that true pneumonia never terminates in gangrene (p. 117) will perhaps be disputed. Of course, pulmonary gangrene must be preceded always by some physical change in the lung, and it is easy to assume, from the presence of dull percussion sound, crepitation, and the like, that inflammation is the preliminary step in every case. Moreover, that pathology which describes gangrene as arising from 'the death of the exudation' necessarily assumes that inflammation always precedes it. If a theory of that kind has to be satisfied, the prevalent notions of what is required to constitute pneumonia are sufficiently elastic to lend it support so far as clinical evidence is concerned, whilst for the rest the circumstance that the gangrenous cavity is found after death surrounded by hepatisation may be adduced as a proof that the gangrene arose upon the pneumonia.

Nevertheless, the cases which are obnoxious to gangrene are precisely those which are least liable to true pneumonia—are those it might almost be said, where the state of the blood renders such an occurrence impossible. We have the testimony of authors that the liability to gangrene varies inversely with the liability to pneumonia. This is believed to be the case with the continued fevers.[1] Gangrene of the lung is more rare in enteric fever than in typhus. Pneumonia is more rare with typhus than with enteric fever.

In works upon chest affections gangrene of the lung is very shortly disposed of; successive writers are content to repeat the accounts which they receive from one another. Dr. Stokes, however, who is little prone to accept doctrines on the mere authority of others, insists upon the close connection between gangrene and what he elsewhere describes as 'typhoid pneumonia.' He adds that in all the cases he has himself seen, the subjects of the gangrene 'had been long addicted to the use of spirits.' Of the four cases whose history he relates, two had received injuries to the chest; the third (who recovered sufficiently to be discharged) was admitted with the symptoms of gangrene fully developed, hæmoptysis having preceded it; and the fourth, a labourer who had fallen drunk into a canal, exhibited in the course of the third day after that accident 'dulness over the right side, without any preceding crepitus.' The day following, the breath and expectoration had all the characters of gangrenous lung. Excluding, then, the cases of injury, what evidence have we here in favour of antecedent pneumonia? The typhoid pneumonia of Dr. Stokes differs indeed in many respects, as he bimself is forward to point out, from simple pneumonia; its connection with gangrene, if it can be made out, will only serve to bring that difference into still stronger relief.

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[1] Murchison, p. 503. See, however, p. 94.

A separate form of gangrene is met with in children; it occurs in several places at once, and the name 'gangræmia' has been applied to it. The affection is often associated with sloughing of the gums and pharynx. It has been supposed by M. Boudet (whose article upon pulmonary gangrene in the 'Archives générales de Médecine,' September 1843, is a valuable monograph upon the subject) that there is a special connection between this gangrene and the poison of measles. It would even seem that it is from this circumstance that gangrene of the lung is more common with children than with adults. Now, the common sequel of measles in weakly children is alveolar catarrh, the so-called 'lobular pneumonia,' and closely corresponding with it is a form of gangrene called by M. Boudet *la forme en noyaux*—small isolated masses, that is, converted in their centres into a putrid detritus.

The initial step in this form of gangrene, therefore, would seem to be pulmonary collapse. It is nearly related to the 'bronchial abscess' described long ago by Dr. Gairdner in connection with collapsed lobules. A tendency towards this termination in gangrene appears to prevail at certain periods with certain epidemic constitutions.

In most cases of 'gangræmia' the general condition is sufficiently manifested by external signs before the state of the lung is such as to excite attention. Gangrene of the gums and mouth precedes the lung symptoms, whilst so soon as these appear their significance is at once made evident by the characteristic odour of the breath.

In the single case of gangrene quoted by Dr. West we have no further proof of antecedent pneumonia than is furnished by a lung which was dull and impervious to air some days before it became obviously fœtid, and Dr. West himself alludes[1] to the absence, in this instance, of those general symptoms which commonly accompany inflammation of the lungs in children.

In regard to other modes of pulmonary gangrene, there are cases tending to show, what *à priori* would appear not improbable, that extravasation of blood into the lung may terminate in a gangrenous cavity, the access of air to a clot determining its putrefaction, and eventually that of the surrounding tissue. Hæmoptysis is often a very prominent symptom of gangrene, both in children and adults. The only case of the affection recorded by Rilliet and Barthez seems to require such an explanation. There is another by Dr. Bristowe ('Pathological Transactions,' vol. viii. p. 57), which might bear the same interpretation. In this instance, where gangrene is ascribed by the author (on insufficient grounds, as I venture to think,) to pneumonia, 'extreme hæmoptysis' had occurred, and constituted, in fact, the chief symptom.

The origin of gangrenous lung from fibrinous deposits in the pulmonary artery was clearly shown by Mr. Callender in a case reported in the ninth volume of the 'Pathological Transactions.' Precisely the same thing was figured by Carswell many years ago (in his Plate III. fig. 4), who describes it as 'a form of mortification occasioned by obliteration of the arteries and veins, in consequence of chronic induration of the pulmonary tissue.' Other examples of a similar kind are not wanting where gangrene of the lung, though ascribed to pneumonia, had been found associated with coagulation of the blood in the pulmonary vessels.[2]

If we add to this list instances of gangrene due to disease of neighbouring parts, such as cancer, carious vertebræ, or the pressure of an aneurismal tumour, we well-nigh exhaust the ascertained causes of this rare form of mortification.

M. Grisolle, who concurs with Frank, Laennec, and Andral in regarding this event of pneumonia as very rare, investigated the histories of seventy reported cases of pulmonary gangrene. He relates that he could hardly find five out of the seventy which could in strictness be regarded as examples of *pneumonia* so terminating. (' De la Pneumonie,' p. 335.)

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[1] West on 'Diseases of Children,' p. 339, fourth edition.

[2] We have seen that pneumonia itself may be found in association with plugged vessels. But for actual observation, therefore, we should be prepared to accept gangrene as one of its accompaniments. The actual fact appears to be that, if not unknown in that relation, it is at least very rare.

Since this book has been in the press, my friend Dr. Thorowgood has given me the particulars of a case in which the characteristic and overpowering fœtor of pulmonary gangrene arose in a pneumonia of no great severity, and which ultimately recovered under his care at the West London Hospital.

I am unable to find upon what evidence gangrene of the lung is associated (as it is by Walshe) with insanity. Its connection with cerebral disease arises, we may suppose, from the dependence of both sometimes upon embolic deposits.

APPENDIX G.

THE STATISTICS, IN REFERENCE TO TREATMENT, OF GRISOLLE, BENNETT, WATERS, PEACOCK, AND OTHERS.

I. GRISOLLE offers a general analysis of 50 cases of *pneumonia treated by bleeding* at the first stage of the disease, and 182 so treated at the second stage. The bleedings were practised from one to five times, upon no uniform plan, more or less often according as the patient responded to it early or late. Hence those that died lost most blood, probably from two to four pounds.

Of the fifty bled at the first stage, five died; of the 182 bled at the second, thirty-two; a mortality much less than Andral's, and in the circumstances of the time and of the cases not unfavourable. M. Grisolle concluded from personal observation that bleeding, however practised and however accompanied, had no power to arrest the disease. He believes that it brought about a general improvement in the patient's condition, but with no corresponding change in his physical state. The benefit to be expected from bleeding is, that it disposes the economy to receive other therapeutic agents. Local bleeding, he believes, has a value of its own in relieving the pain of stitch.

The value of *tartar emetic* was tried by the same observer upon 154 persons, grouped as follows: forty-four were treated with it alone in half-hourly or hourly *emetic* doses; eighty (after the manner of Laennec) were bled first and given antimony subsequently and in place of further bleeding; the remaining thirty were given antimony as a *pis aller* at a late period of their illness and because further bleeding was deemed impracticable. Of the first group Grisolle lost six, or one in 7⅓, but *none of these commenced the treatment till after the fifth day.* Of the second group ten died, or one in eight; the treatment with them was not begun till the sixth or seventh day. Of the third group eighteen died out of the thirty. Of the twelve who recovered, nine 'in the greatest peril' improved rapidly under the antimony. Of the whole 15 only twelve absolutely 'tolerated' the remedy, yet in all 'it was evident from the diminished *malaise* that it should be persisted with.' The patients improved with a rapidity unobserved in any other treatment (a statement corroborated, as we have seen, by Trousseau).

Yet the effect of the antimony was not in itself pleasing. Not rarely the drug produced in the first twenty-four hours from ten to fifteen vomitings and from twenty to thirty stools, the latter being always in excess of the former; the treatment was, indeed, regulated to produce such effects. Yet upon the concurrent testimony of two independent observers, the general improvement was so obvious as to suggest the figure of a man snatched from the grave.[1]

The rates of mortality from pneumonia of the most eminent of the French physicians are usually stated as follows, though the value of such statement may well be questioned.

Louis	30·8 per cent.
Andral	55·4 ,,
Chomel	32 ,,
Bouillaud	11 or 12 ,,

Rasori's tartar emetic and bleeding treatment gave 22 per cent.

II. The statistics of Dr. Hughes Bennett will be found in his paper, 'The Restorative Treatment of Pneumonia,' published in 1865. It consists of a table of cases, together with a careful analysis, a statistical review of various systems of treatment, and answers to various objectors. The pamphlet must be read; I cannot pretend to do justice to it. It is only necessary here to verify the statement of the text (Chap. xiv., p. 213), that the worst cases were treated according to their supposed needs, apart, so far as appears, from any speculative views, and that Dr. Bennett's success is in part attributable to the fact that he does not permit his action to be too closely fettered by his argument.

It is to be understood that the 129 cases were treated consecutively, i.e. *all* the examples of pneumonia are included, admitted into the Edinburgh Royal Infirmary between 1848 and 1861. It follows, of course, that many of these (it may be doubtful how many) are cases of no severity. I claim at least to have included in the list following (*which is a verbatim extract*) nearly all the crucial cases, and I ask what particular treatment do they illustrate?

---

[1] Grisolle, loc. cit. p. 634. I am reminded to observe here, for what it is worth, that the most extreme pneumonia I ever saw recover, was treated at St. George's Hospital by Dr. Pitman, with antimony in emetic doses.

No.		First seen after rigor	Pulse	Respirations	Treatment	Observations
		days				
MEN	21	5	130	30	¼ grain antimony every hour, afterwards every two hours.	Rapid recovery.
	22	5	148	56	Antimony ⅙ grain every three hours, afterwards nutrients.	Convalescence not determined.
	24	6	120	44	⅓ grain antimony every two hours, diuretics ʒvj., wine, and nutrients.	Of intemperate habits.
	36	7	120	40	Salines, diurctics, c. colchicum wine ℥iv., and nutrients.	A debilitated intemperate man.
	37	1	120	36	Salines, blister, nutrients.	A case of pleurisy, pneumonia intercurring.
	48	6	120	48	Wine ℥iv. daily, liquid nutrients *ad lib.*, slight salines.	Recovered rapidly.
	51	2	120	46	Salines, strong beef-tea, wine ℥iv.	
	61	5	104	56	Salines, slight diuretics, wine ℥vi., nutrients.	Very severe case.
	75	3	120	66	Nutrients, wine ℥vj.	Rapid recovery.
WOMEN	86	6	120	dyspnœa	Bled to ℥xij. on admission, ½ grain antimony every two hours.	Record defective.
	89	8	132	hurried	Digitalis, laxatives, 3 leeches to side.	Great exhaustion and unusual action of heart.
	95	5	120	hurried	1 grain antimony every two hours, afterwards 12 leeches, wine ℥vj., blisters.	Very weak after subsidence of fever.
	96	1	120	36	Salines, 8 leeches, and aftcrwards blistcrs.	A simple pneumonia.
	105	7	120	2	Antimony ⅓ grain and diuretics.	

	No.	First seen after rigor	Pulse	Respirations	Treatment	Observations
		days				
WOMEN	106	2	130	32 to 36	Antimony ⅓ grain every third hour, after ¼ grain every fourth hour.	
	107	5	112	40	⅓ grain antimony, 10 leeches, and a blister.	
	111	11	120	urgent dyspnœa	Wine ; nutrients ? ℥vij. (sic).	
	114	1	120	dyspnœa	Salines, nutrients, wine ℥iv.	Complicated with bronchitis and phthisis.
	116	3	150	56	At first salines, afterwards diuretics, ℥ss. wine every half-hour, new milk, and strong beef-tea.	*Saved by ℥ss. wine every half-hour.*
	124	8	120	65	Salines, wine ℥iv., nutrients.	

These are, it is true, but twenty cases out of more than a hundred, but for testing the point at issue they are not to be estimated numerically. They are *the* cases upon which the efficacy of the treatment is to be tested. And what is that treatment ? With no less than eight of the twenty it is antimony ; with the others it is wine, which is given in larger or smaller quantity according to individual need, sometimes in aid of the 'nutrients,' and sometimes for the immediate saving of life.

III. Dr. Waters tabulates seventy-seven cases. From a careful analysis of these I learn that at least twenty-three were admitted on or after the eighth day. Fifty-four only can be taken as fair illustrations of the hospital treatment. Of these twenty-six, as far as can be judged from the details given, were serious cases. One died. Of cases of simple pneumonia, subjected to Dr. Waters' treatment within a week of commencing illness, I cannot reckon more than twenty-three. Of these nine were severe. Three of the number were treated with antimony, and six with wine or brandy. (See Dr. Waters 'On Diseases of the Chest,' article 'Pneumonia.')

IV. *Tabular Statement of Results of Treatment in Pneumonia* (*from Dr. Rogers' 'Therapeutics.'*)

	Where and by whom treated	No. of patients	No. of deaths	Average mortality
	Homœopathic Treatment—			
	Fleischmann (7 successive years)	239	14	5·85
	Eidherr (7 successive years)	...	...	7·22
	Homœopathic section of Leopoldstadt Hospital (6 successive years)	94	9	9·57
	Do. Wurmb and Caspar, 1850	24	3	12·55
	Tessier	41	3	7·3
	Non-Homœopathic—			
1st Group	Hegelé, hydropathic treatment	40	...	...
	Barthez, expectant treatment	212	2	0·94
	Ziemssen ,, ,,	201	7	3·48
	Dietl (1st series) expectant treatment	189	14	7·4
	,, (2nd series) ,, ,,	750	69	9·2
2nd Group	Bennett, restorative treatment	129	4	3·1
	Kissel (acet. ferri or acet. cupri)	112	5	4·4
	Sauer (sulph. cupri)	56	3	5·35
	Edinburgh Infirmary (1865)	36	3	8·33
	Allopathic section of Leopoldstadt Hospital (6 successive years)	104	13	12·50
	General Hospital Vienna (10 successive years)	...	...	18·28
	Huss* (8 successive years) 2nd series	...	...	10·21
3rd Group	Lebert	...	...	7·3
	Huss (8 successive years) 1st series	...	...	11·50
	Grisolle†	...	...	16
	Louis	...	...	30

The groups are defined as follows :—

1st Group.—That in which no bleeding, and few or no drugs were employed.

2nd Group.—That in which medicine was more employed, and bleeding sparingly.

3rd Group.—That in which both medicine and bleeding were actively employed.

V. To the statistics of Dr. Peacock, recently published in the fifth volume of the 'St. Thomas's Hospital Reports,' I can but draw the reader's attention. As a contribution to the clinical records upon the subject, this summary has been rarely equalled in its completeness of detailed analysis. The numbers dealt with are not large, and the rate of mortality (which is reckoned at 11·7) is therefore unimportant. Dr. Peacock remarks upon the large proportion of cases admitted in the summer. He calls attention also to the large number of his own cases where the pneumonia was double. Of twenty-six at the Royal Free Hospital eleven were double; that is to say (excluding thirteen where the precise seat was not named), nearly one-half. Of fifty-eight at the St. Thomas's Hospital both sides were affected in twenty-five, the right in seventeen, the left in sixteen. This large proportion of double pneumonia is quite exceptional, and suggests that different physicians restrict the disease differently.

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* Huss's statistics extend from 1840 to 1855, and include 2,616 cases, with a total mortality of 281, or 10·74 per cent., the largest numbers we have to deal with, as I believe. The yearly rates, however, are very various. In 1843, 14·19 per cent. died, in 1851, only 6·19; an instructive observation in reference to the conclusions to be drawn from statistics.

† Grisolle's statistics, as I have noticed already, cannot fairly be stated in this summary manner.

Of the 'complicated cases,' exception might be taken to the inclusion of 'violent delirium' in that category, or 'delirium and diarrhœa,' or even 'hæmoptysis,' unless profuse. These are but instances of the undue prominence of a particular symptom, itself not foreign to the disease. It will be observed, as might be expected, that the double pneumonias are 'complicated' in much larger proportion than the single.

In regard to temperature Dr. Peacock states that it is 'undoubtedly the best measure of intensity' (p. 12). Of thirty-two cases, three only exceed (by a little) 104°. A fatal case of double pneumonia (proved to be simple by post-mortem examination) reaches its highest temperature, 102·5°, on the 4th day, and then, as with a recovering crisis, falls continuously until death on the 6th day. A case with a very high temperature 'not corresponding with the general character of the attack' Dr. Peacock believes on that account to be an error in observation, and therefore omits it. For myself I should be disposed to accept the record as being in accordance with what is not rarely seen as well in pneumonia as in typhoid fever.

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LONDON : PRINTED BY
SPOTTISWOODE AND CO., NEW-STREET SQUARE
AND PARLIAMENT STREET